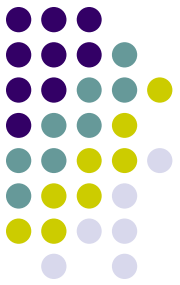
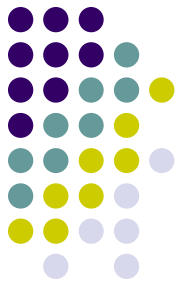


# Dry Eye Syndrome



Before you begin: This is a big topic, and big topics beget big slide-sets. There are natural breaks at slides 166, 276, 427, 482, and 654; I placed a *break time!* slide at those points to mark them.

# Dry Eye Syndrome



*How common is DES?*

## Dry Eye Syndrome



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Very. It is estimated to affect

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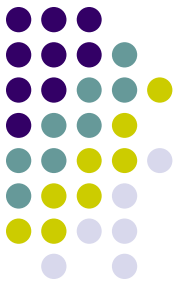
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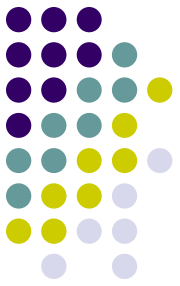
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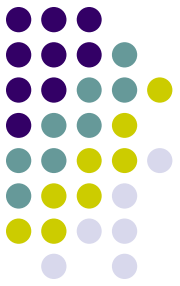
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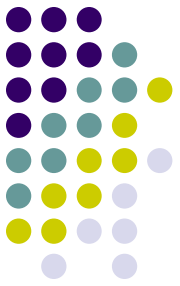
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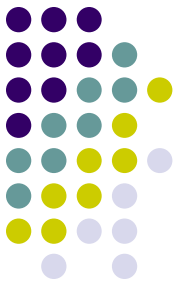
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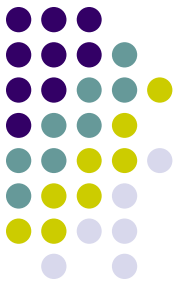
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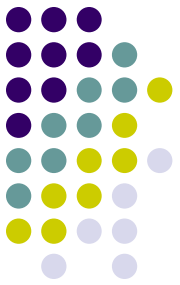
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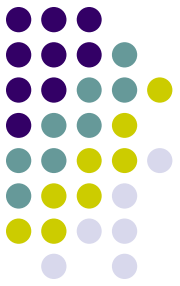
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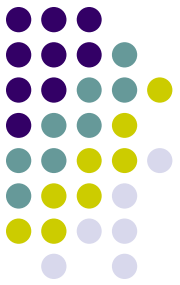
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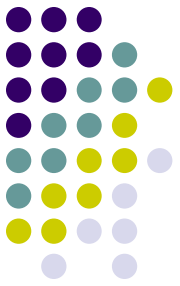
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...women are more likely to have DES if they are receiving **two words** therapy

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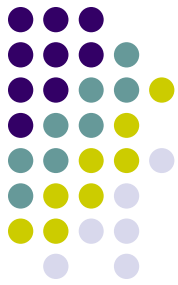
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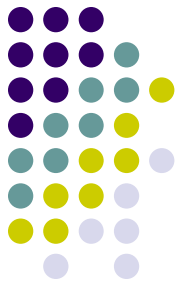
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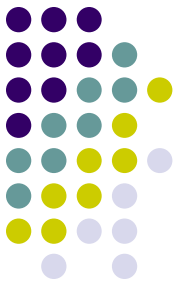
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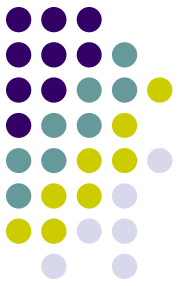
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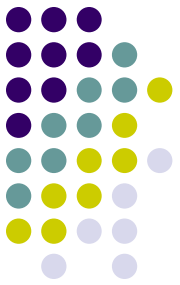
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# Dry Eye Syndrome

*What roles does the tear film play in ocular health and function?*



# Dry Eye Syndrome

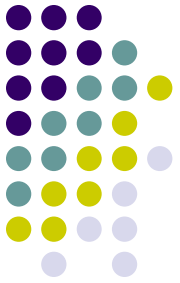
*What roles does the tear film play in ocular health and function?*

There are three:

--?

--?

--?





# Dry Eye Syndrome

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## Dry Eye Syndrome

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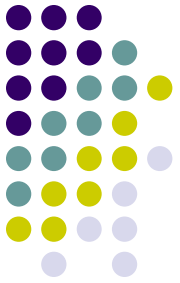
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## Dry Eye Syndrome



*What roles does the tear film play in ocular health and function?*

There are three:

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- Assists in clearing debris from the corneal surface
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## Dry Eye Syndrome



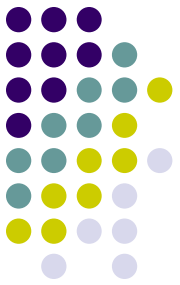
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## Dry Eye Syndrome



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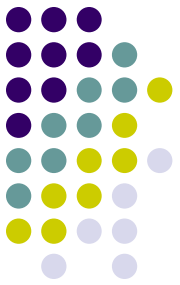
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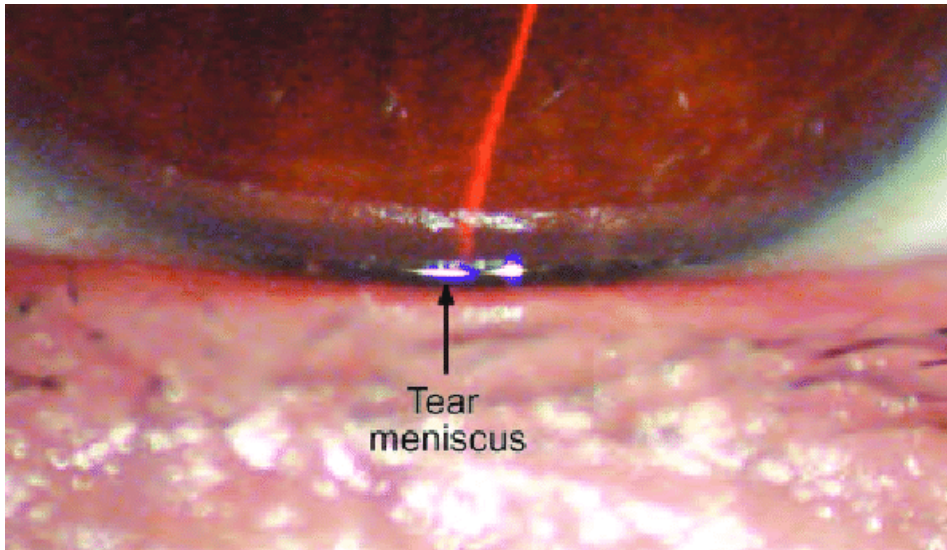
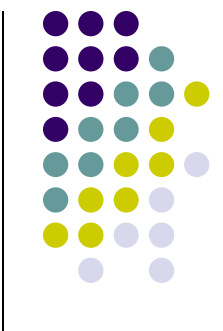
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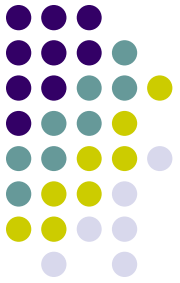
# Dry Eye Syndrome



Tear lake (strip; meniscus)



## Dry Eye Syndrome



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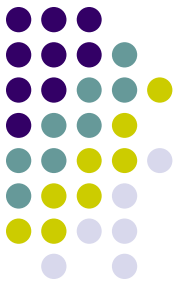
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The bulk of tear volume is in the tear strip or lake (aka the tear *meniscus* ) resting on the lower-lid margin

*How does the tear volume get from the tear strip up onto the ocular surface where it's needed?*

Courtesy of the action of the upper lid (UL) . During a blink, the UL travels down across most of the extent of the two words (not 'ocular surface')- (the lower lid goes up a little, but not much).

## Dry Eye Syndrome



*What roles does the tear film play in ocular health and function?*

There are three:

- Facilitates diffusion of oxygen to the avascular cornea
- Assists in clearing debris from the corneal surface
- Provides a glassy-smooth refracting surface at the air-cornea interface (or more accurately, the air-tear film interface)

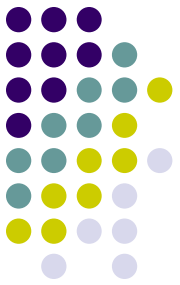
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Courtesy of the action of the upper lid (UL) . During a blink, the UL travels down across most of the extent of the interpalpebral fissure (the lower lid goes up a little, but not much).

## Dry Eye Syndrome



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Courtesy of the action of the upper lid (UL) . During a blink, the UL travels down across most of the extent of the interpalpebral fissure (the lower lid goes up a little, but not much). As it goes down the UL wipes debris off the surface and into the lake. As it goes back up, the UL exerts a capillary-attraction force on the aqueous in the tear lake, thereby pulling it up across the ocular surface. (The oil layer follows along.)

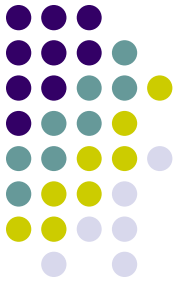
## Dry Eye Syndrome

*The tear film is comprised of # basic components.*



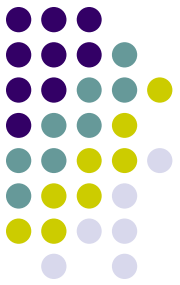
# Dry Eye Syndrome

*The tear film is comprised of three basic components.*





## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

--?

--?

--?

## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

- Lipid
- Aqueous
- Mucin

## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

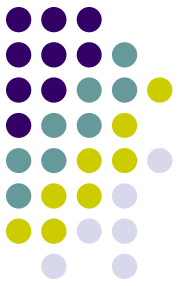
--Lipid

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*How are the three components physically related to one another?*

## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

--Lipid

--Aqueous

--Mucin

*How are the three components physically related to one another?*

The aqueous and mucus components are intermixed into a single, gel-like layer (the '  phase'), which in turn is covered by a lipid layer

## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

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## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

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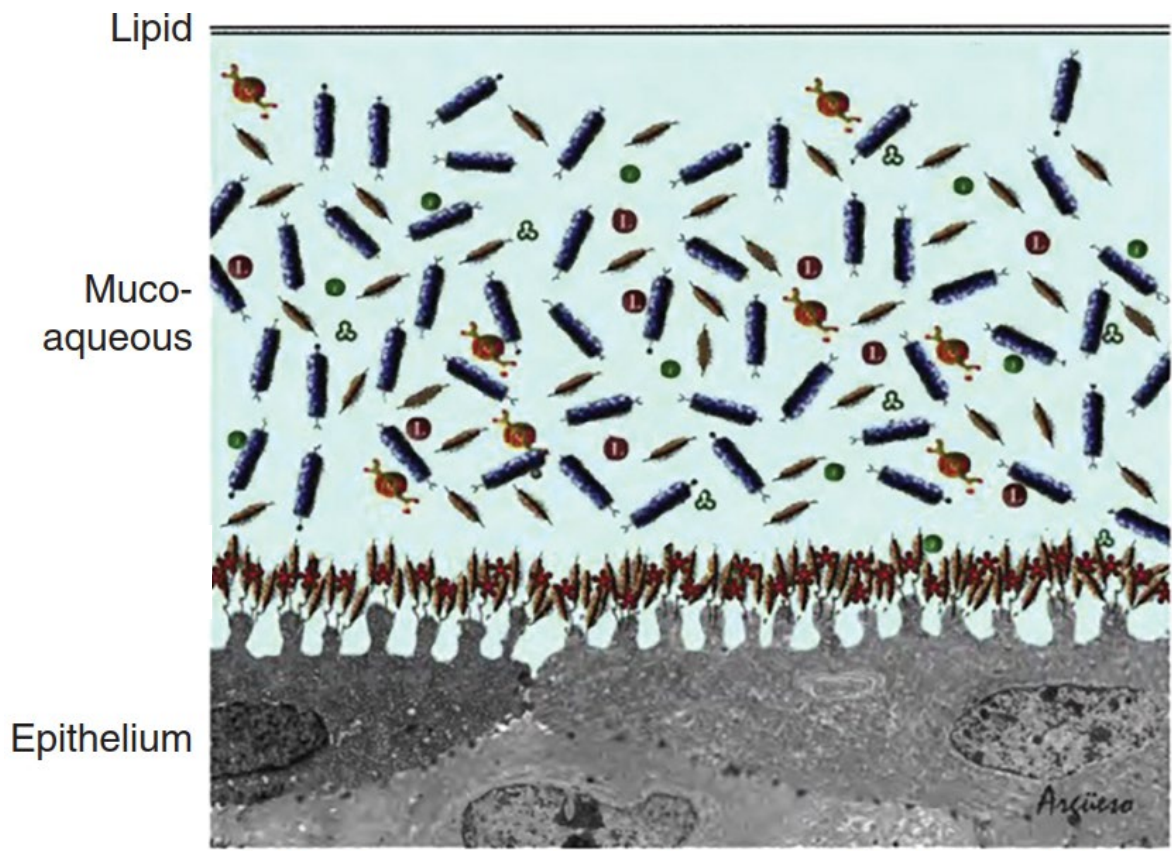
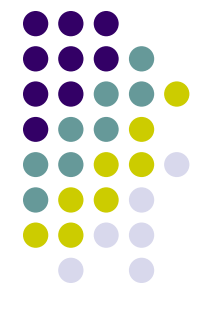
--Aqueous

--Mucin

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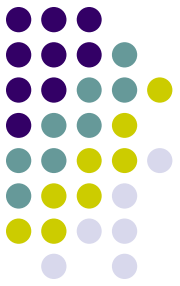
The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer. *This is the two-phase model of the tear film.*

# Dry Eye Syndrome



Two-phase model of the tear film. Schematic drawing of the structure of the tear film showing the outer lipid layer and the mucoaqueous layer.

## Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

--Lipid

--Aqueous

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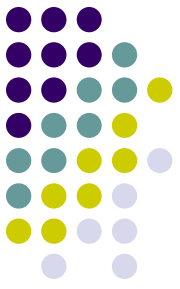
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*As an aside: Briefly, what is the tripartite model of the tear film?*



## Dry Eye Syndrome



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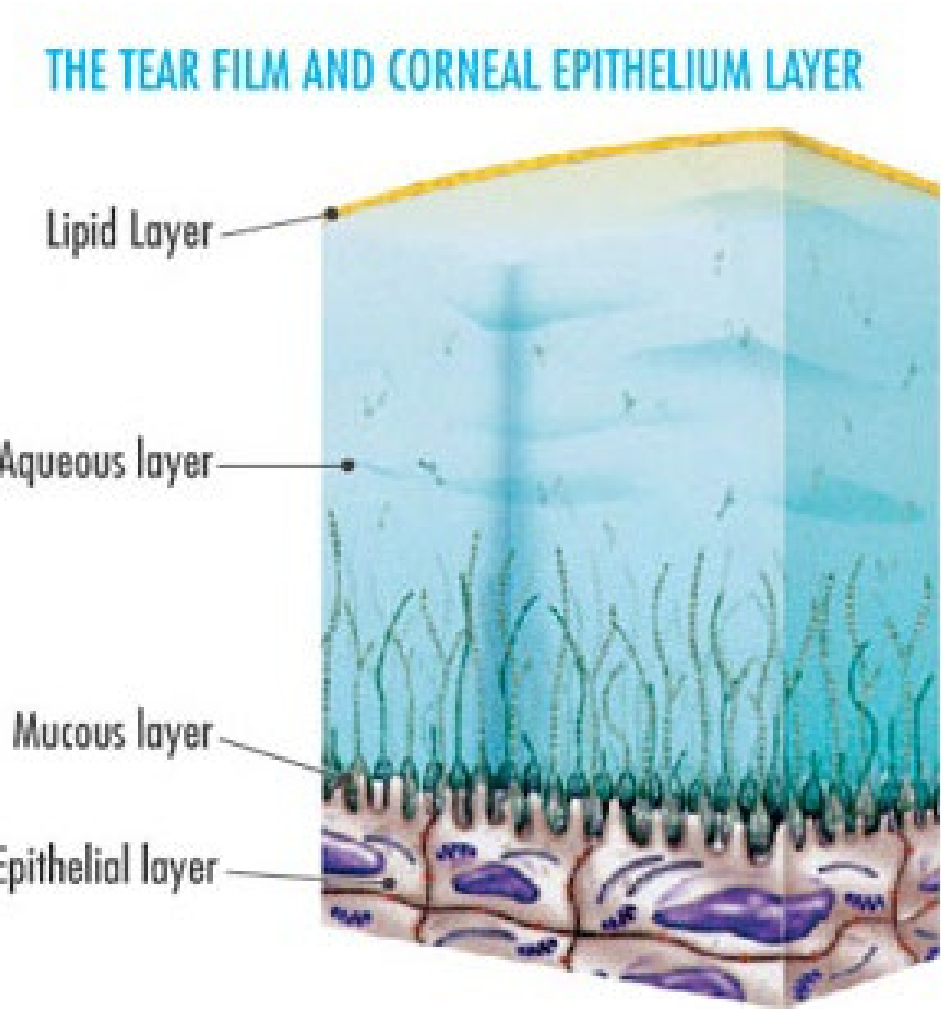
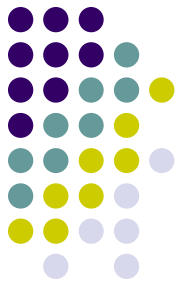
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*As an aside: Briefly, what is the tripartite model of the tear film?*

The idea that the tear film is composed of *three* separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers

# Dry Eye Syndrome



The *tripartite model* of the tear film

## Dry Eye Syndrome



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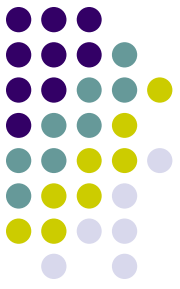
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## Dry Eye Syndrome



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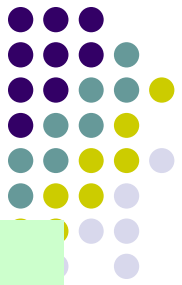
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While once widely accepted, consensus now is it’s incorrect

# Dry Eye Syndrome



The tear film

**Lipid**

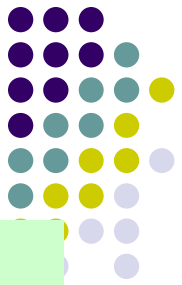
--Aqueous  
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The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?  
--?  
--?  
  
How are they formed?  
--?  
  
The aqueous layer is a gel-like layer, and the lipid layer is a lipid layer.

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# Dry Eye Syndrome



The tear film  
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--Aqueous  
--Mucin

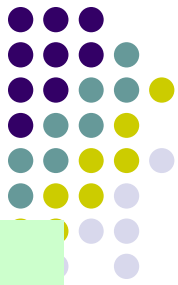
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# Dry Eye Syndrome



The tear film  
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**Lipid**

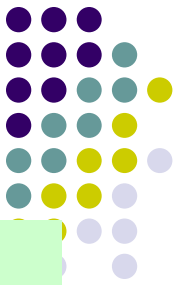
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# Dry Eye Syndrome



## Lipid

The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

--Inhibit tear film evaporation , thereby keeping it on the eye longer

--Reduce tear film two words , thereby keeping it on the eye longer

How are they --?

The aqueous  
gel-like layer  
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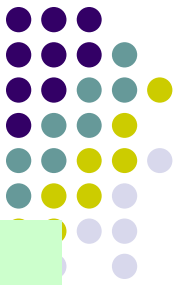
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# Dry Eye Syndrome



The tear film

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--Inhibit tear film evaporation , thereby keeping it on the eye longer

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How are they

--?

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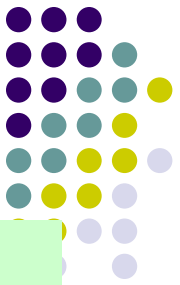
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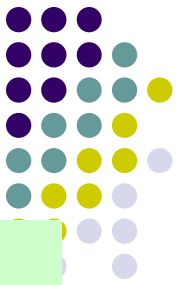
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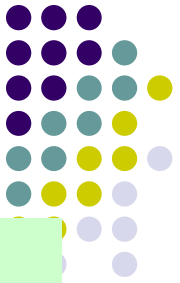
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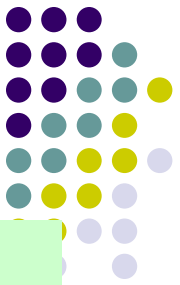
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# Dry Eye Syndrome



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*Which gland(s) produce the lipids constituting this layer?*

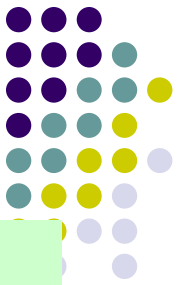
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*Which gland(s) produce the lipids constituting this layer?*

The meibomian glands

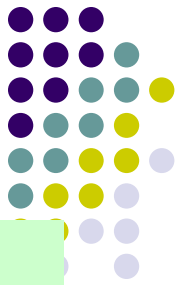
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# Dry Eye Syndrome



The tear film consists of three layers:  
--Aqueous  
--Mucin

**Lipid**

The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

- Inhibit tear film evaporation, thereby keeping it on the eye longer
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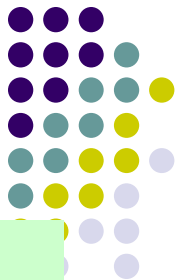
Which gland(s) produce the lipids constituting this layer?  
The **meibomian glands**

As an aside...  
The idea of the tripartite model is that the tear film consists of three layers: aqueous, mucin, and lipid layers.

The meibomian glands are embedded within the **specific structure**

There is a problem with the tripartite model—what is it?  
While once widely accepted, consensus now is it's incorrect

# Dry Eye Syndrome



The tear film

**Lipid**

--Aqueous  
--Mucin

The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

- Inhibit tear film evaporation, thereby keeping it on the eye longer
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- Without a lipid layer, surface tension (along with gravity) would pull the tear film down the eye to the lake
- Facilitate visual acuity by providing a smooth refracting surface

How are they

The aqueous  
gel-like layer  
a lipid layer

Which gland(s) produce the lipids constituting this layer?

The **meibomian glands**

As an aside

The idea  
layers each  
aqueous, and lipid layers

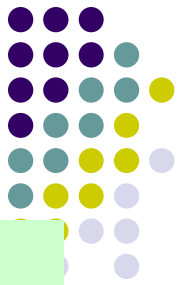
The meibomian glands are embedded within the **tarsal plates**

There is a problem with the tripartite model—what is it?

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# Dry Eye Syndrome



The tear film

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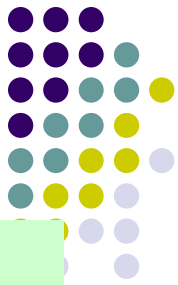
The meibomian glands are embedded within the **tarsal plates**

Upper lid, lower lid, or both?

There is a problem with the tripartite model—what is it?

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# Dry Eye Syndrome



The tear film consists of three layers:  
--Aqueous  
--Mucin

**Lipid**

The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

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- Facilitate visual acuity by providing a smooth refracting surface

Which gland(s) produce the lipids constituting this layer?  
The **meibomian glands**

How are the lipids secreted?  
The aqueous humor is a gel-like substance that contains a lipid layer.

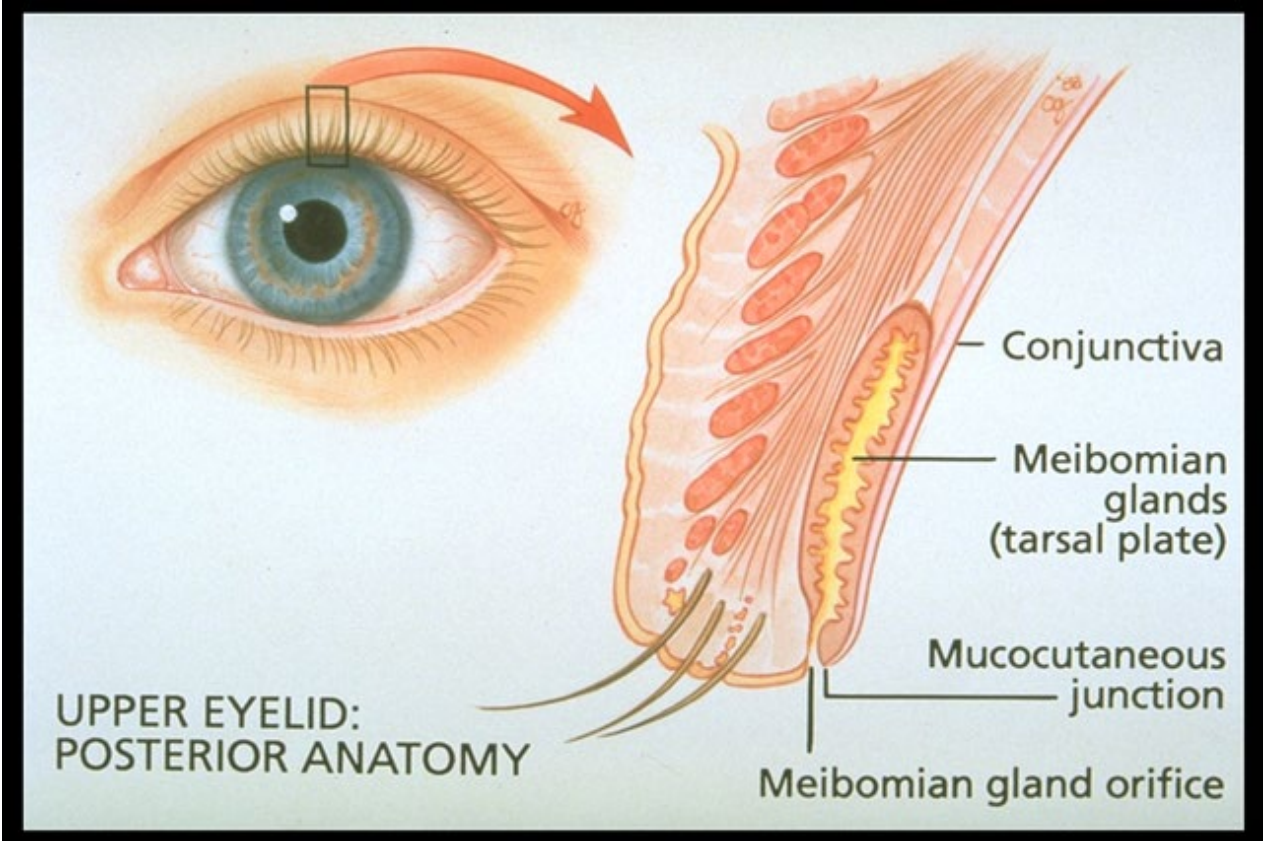
As an aside, the meibomian glands are embedded within the tarsal plates. The idea is that the lipid layers each have an aqueous, mucin, and lipid layer.

The meibomian glands are embedded within the **tarsal plates**.

Upper lid, lower lid, or both?  
Both

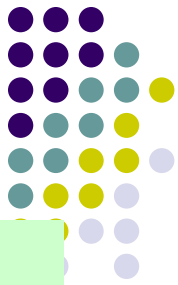
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# Dry Eye Syndrome



Meibomian glands

# Dry Eye Syndrome



The tear film consists of three layers:

- Lipid**
- Aqueous
- Mucin

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How are the lipids produced?

The aqueous layer is a gel-like layer and the lipid layer is a lipid layer

Which gland(s) produce the lipids constituting this layer?

The **meibomian glands**

As an aside...

The idea of the three layers each having their own aqueous, lipid, and mucin layers

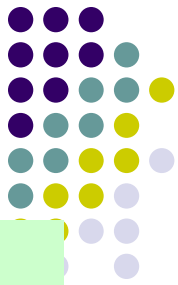
The meibomian glands are embedded within the **tarsal plates**

The product of a meibomian gland is called **duh**

There is a problem with the tripartite model—what is it?

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# Dry Eye Syndrome



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Which gland(s) produce the lipids constituting this layer?  
The **meibomian glands**

As an aside:  
The idea of the three layers each having its own aqueous, mucin, and lipid layers is

The meibomian glands are embedded within the **tarsal plates**  
The product of a meibomian gland is called **meibum**

There is a problem with the tripartite model—what is it?  
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# Dry Eye Syndrome



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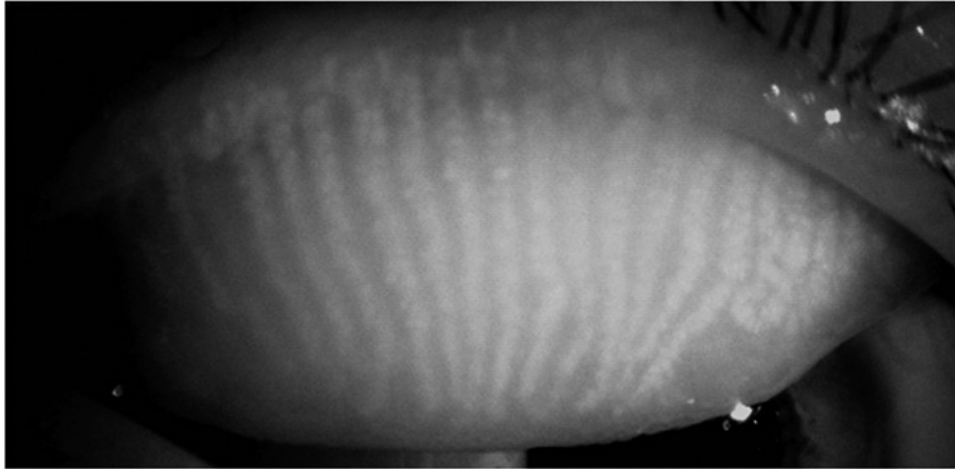
The meibomian glands are embedded within the **tarsal plates**  
The product of a meibomian gland is called **meibum**  
There are up to twice as many meibomian glands in the **upper** lids

There is a problem with the tripartite model—what is it?  
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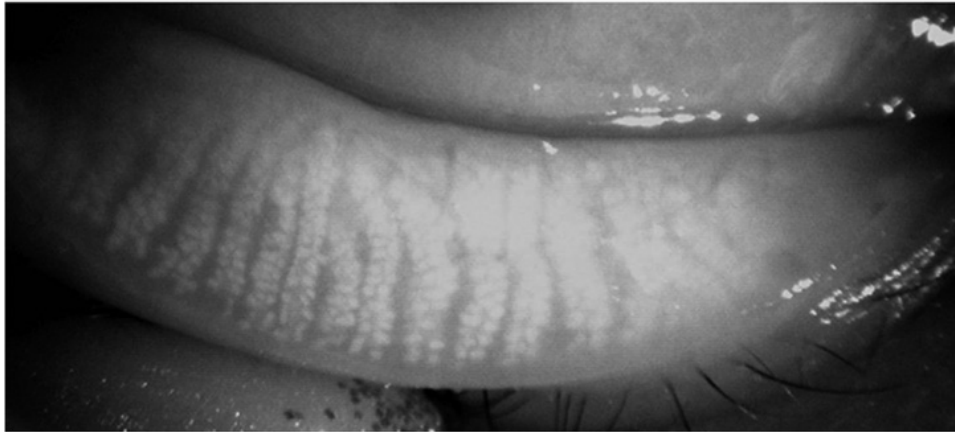
# Dry Eye Syndrome



Upper lid



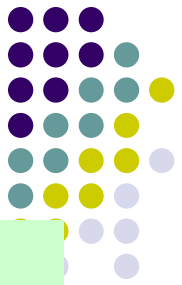
Lower lid



Meibomian glands



# Dry Eye Syndrome



The tear film consists of three layers:

**Lipid**

--Aqueous  
--Mucin

The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

- Inhibit tear film evaporation, thereby keeping it on the eye longer
- Reduce tear film surface tension, thereby keeping it on the eye longer
- Without a lipid layer, surface tension (along with gravity) would pull the tear film down the eye to the lake
- Facilitate visual acuity by providing a smooth refracting surface

How are lipids produced?

The aqueous humor is a gel-like substance that contains a lipid layer.

Which gland(s) produce the lipids constituting this layer?

The **meibomian glands**

As an aside...

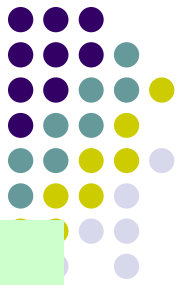
The idea of the lipid layer is that each layer has its own aqueous, mucin, and lipid layers.

The meibomian glands are embedded within the **tarsal plates**.  
The product of a meibomian gland is called **meibum**.  
There are up to twice as many meibomian glands in the **upper lids**.  
The meibomian glands are innervated primarily by the **sympathetic? parasympathetic? somatic?** system.

There is a problem with the tripartite model—what is it?

While once widely accepted, consensus now is it's incorrect.

# Dry Eye Syndrome



The tear film consists of three layers:  
--Aqueous  
--Mucin

**Lipid**

The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

- Inhibit tear film evaporation, thereby keeping it on the eye longer
- Reduce tear film surface tension, thereby keeping it on the eye longer
- Without a lipid layer, surface tension (along with gravity) would pull the tear film down the eye to the lake
- Facilitate visual acuity by providing a smooth refracting surface

Which gland(s) produce the lipids constituting this layer?  
The **meibomian glands**

How are the lipids secreted?  
The aqueous humor is a gel-like substance that contains a lipid layer.

As an assistant, I can help you with your question. The idea is that the lipid layers each have a different function: aqueous, mucin, and lipid layers.

The meibomian glands are embedded within the **tarsal plates**  
The product of a meibomian gland is called **meibum**  
There are up to twice as many meibomian glands in the **upper lids**  
The meibomian glands are innervated primarily by the **parasympathetic** system

There is a problem with the tripartite model—what is it?  
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# Dry Eye Syndrome



The tear film

--Lipid

--Aqueous

--Mucin

What gland-type secretes the aqueous portion of the tear film?

How are the

The aqueous

gel-like layer

a lipid layer

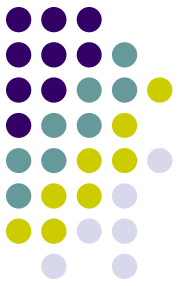
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The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers

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Lacrimal gland

How are the

The aqueous

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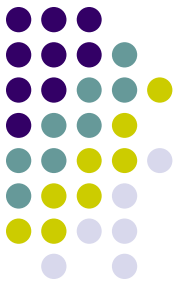
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The tear film

--Lipid

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What gland-type secretes the aqueous portion of the tear film? ?

Lacrimal gland

How many lacrimal glands are there (in each orbit)?

How are the

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Lacrimal gland

How many lacrimal glands are there (in each orbit)?

Lots! But we think of them as being in one of two groups:

--?

--?

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--The [redacted] lacrimal gland

--The [redacted] lacrimal glands

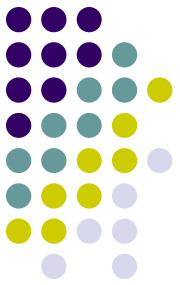
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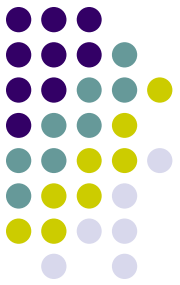
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*Are they innervated?*

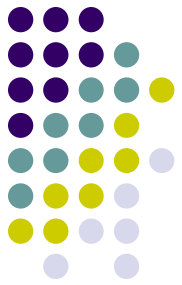
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Yes, primarily by nerves of the

sympathetic?  
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*Are they innervated?*

Yes, primarily by nerves of the parasympathetic system

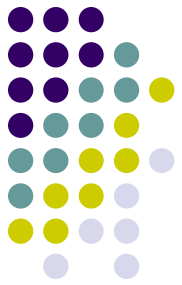
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Where is the main lacrimal gland located?

The superotemporal orbit

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The tear film

--Lipid

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What gland-type secretes the aqueous portion of the tear film?

Lacrimal gland

How many lacrimal glands are there (in each orbit)?

Two! But we think of them as being in one of two groups:

--**The main lacrimal gland**

--The accessory lacrimal glands

How are the

The aqueous

gel-like layer

a lipid layer

Are they innervated?

Yes, primarily by nerves

Where is the main lacrimal gland located?

The superotemporal orbit

It's divided into two lobes—what are they called?

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The aqueous

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Are they innervated?

Yes, primarily by nerves

Where is the main lacrimal gland located?

The superotemporal orbit

It's divided into two lobes—what are they called?

The orbital and palpebral lobes

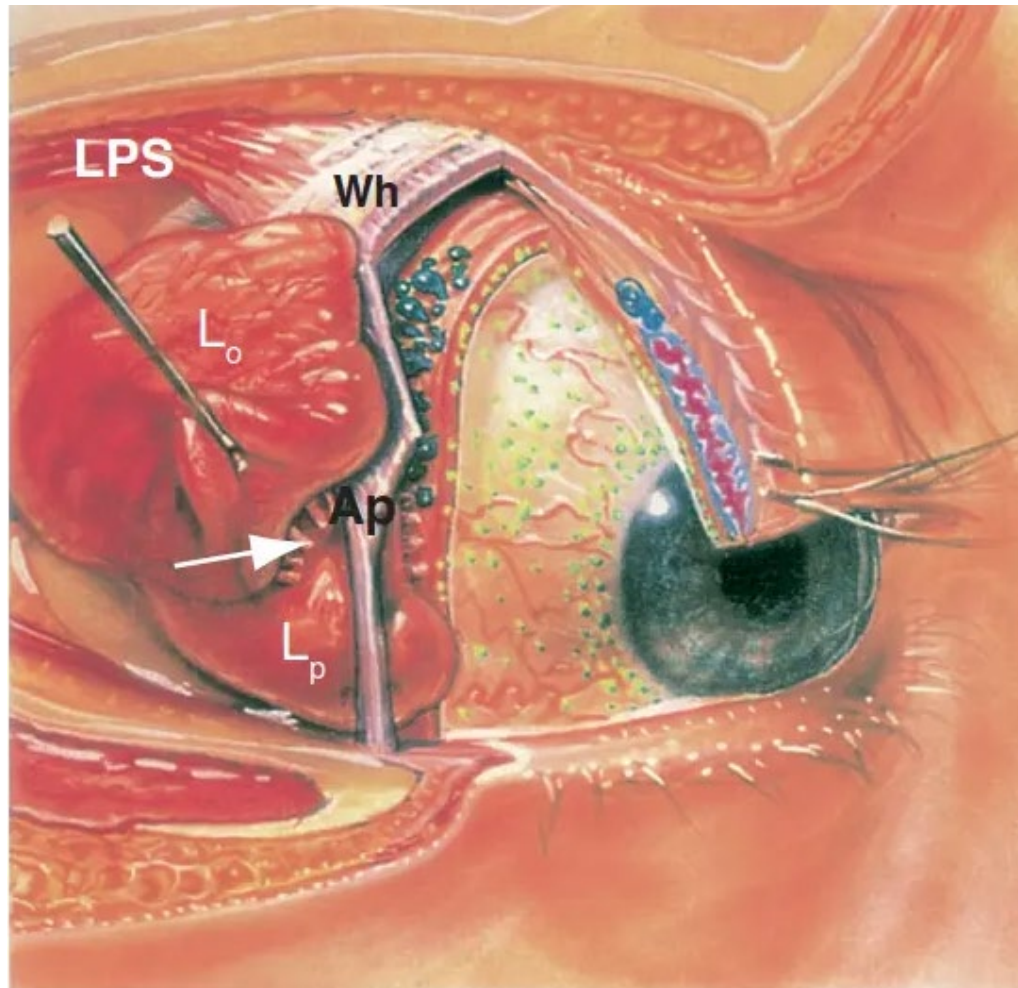
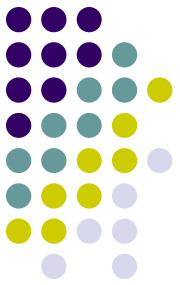
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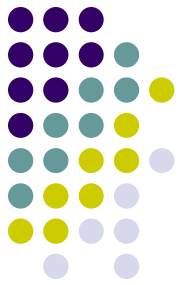
## Dry Eye Syndrome



The orbital lobe of the lacrimal gland (L<sub>o</sub>) and the palpebral lobe of the lacrimal gland (L<sub>p</sub>) are separated by the lateral horn of the levator aponeurosis (Ap) (FYI: LPS = levator palpebrae superioris; Wh = Whitnall's ligament)



# Dry Eye Syndrome



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How are the

The aqueous

gel-like

a lipid

There are two eponymous accessory glands—what are they?

--Glands of

--Glands of

As an

The ide

layers

aqueous,

system

film?

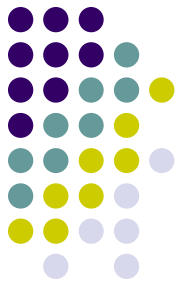
and distinct

ucus,

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# Dry Eye Syndrome



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--Lipid  
Lacrimal gland

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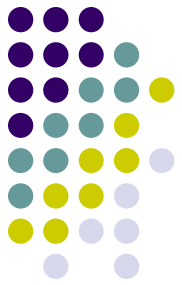
gel-like There are two eponymous accessory glands—what are they?

- Glands of Krauss
- Glands of Wolfring

As an e... film?  
The ide... and distinct  
layers... ucus,  
aqueous, ...

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What is the primary location for each?

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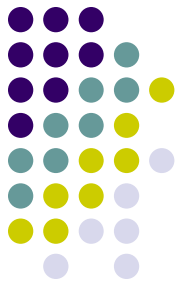
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Are these large, singular structures a la the main lac gland?

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Are these large, singular structures a la the main lac gland?

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No, they are two sets of (much smaller) glands distributed throughout the orbit

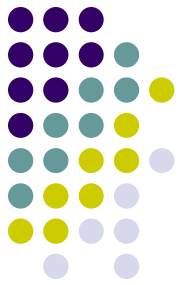
aqueous

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# Dry Eye Syndrome



The tear film *What gland-type secretes the aqueous portion of the tear film?*

--Lipid

**Lacrimal gland**

**Aqueous**

--Mucin

*In addition to secreting its aqueous component, the lacrimal glands contribute important 'microconstituents' of the tear film. What are these?*

How are the *--?*

The aqueous *--?*

gel-like *--?*

a lipid

*There are two types of accessory glands: meibomian glands and glands of Krauss and Wolfring.*

*What is the primary location for each?*

--Glands of Krauss, found in the fornices

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As an

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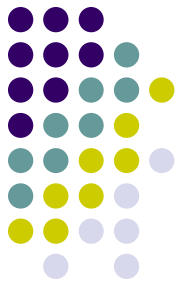
layers

*aqueous, mucin, and lipid*

*There is a problem with the tripartite model—what is it?*

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How are the

--Electrolytes

The aqueous

--Solutes

gel-like

--Proteins

a lipid

*There are two types of accessory lacrimal glands. What is the primary location for each?*

--Glands of Krauss, found in the fornices

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As an

system

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aqueous,

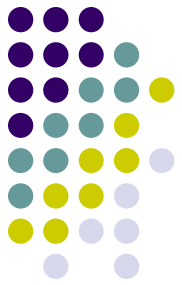
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# Dry Eye Syndrome



The tear film

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How are the

**Electrolytes**

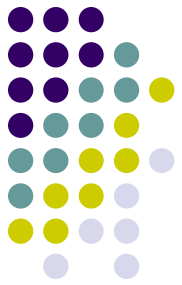
The aqueous

--Solutes

What is the primary role of electrolytes in the tear film?

There is a problem with the tripartite model—what is it?  
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# Dry Eye Syndrome



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--Aqueous

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To regulate tear-film

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**Electrolytes**

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What is the primary role of electrolytes in the tear film?

To regulate tear-film osmolarity

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What gland-type secretes the aqueous portion of the tear film?

Lacrimal gland

In addition to secreting its aqueous component, the lacrimal glands contribute important 'microconstituents' of the tear film. What are these?

--Electrolytes

--Solute

How are the

The aqueous

What is the primary role of electrolytes in the tear film?

To regulate tear-film osmolarity

In a sentence or two, what is osmolarity?

There

While once widely accepted, consensus now is it's incorrect

# Dry Eye Syndrome



The tear film *What gland-type secretes the aqueous portion of the tear film?*

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To regulate tear-film

**osmolarity**

*In a sentence or two, what is osmolarity?*  
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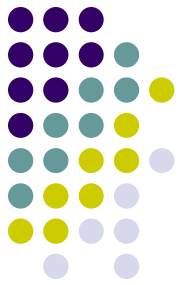
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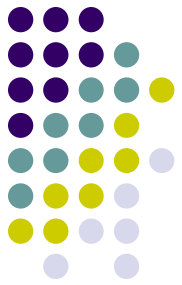
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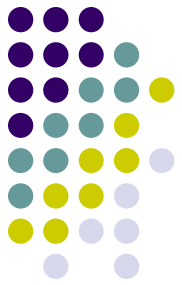
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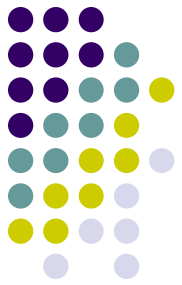
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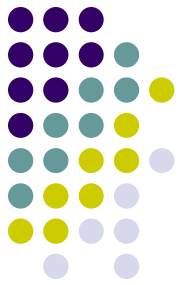
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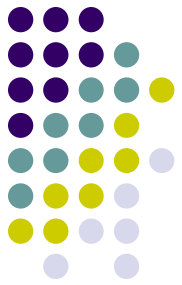
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\*This is from EyeWiki, not the BCSC. Caveat emptor.

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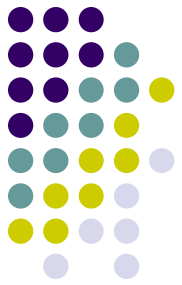
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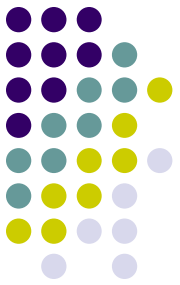
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gel-like layer

a lipid layer

Yes, primary

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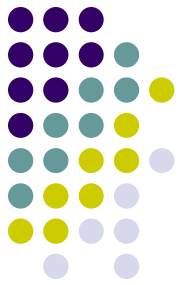
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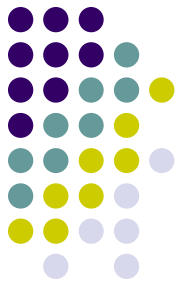
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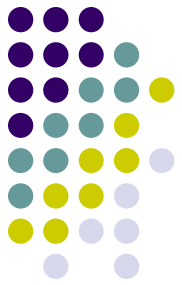
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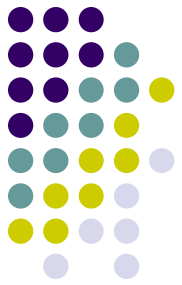
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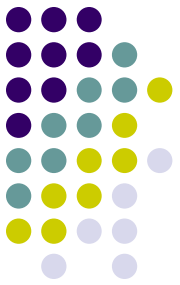
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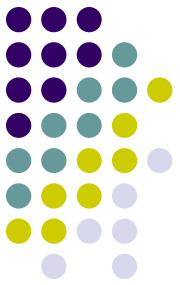
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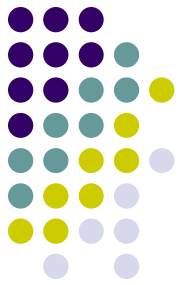
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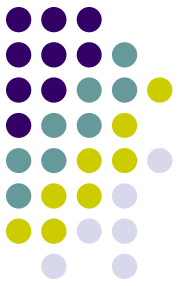
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Goblet cells

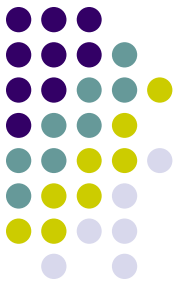
*As an aside: Briefly, what is the tripartite model of the tear film?*

The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers

*There is a problem with the tripartite model—what is it?*

While once widely accepted, consensus now is it's incorrect

# Dry Eye Syndrome



*The tear film is comprised of three basic components. What are they?*

--Lipid

--Aqueous

--Mucin

*What is the chief function of the mucin component of the mucoaqueous layer?*

Facilitating surface wetting by transforming the epithelial surface from a hydrophobic to a hydrophilic state

*How are*

*The aqueous*

*gel-like layer*

*a lipid layer*

*Which cells are the chief producers of mucins?*

Goblet cells, which are found in the                      epithelium

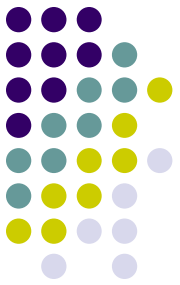
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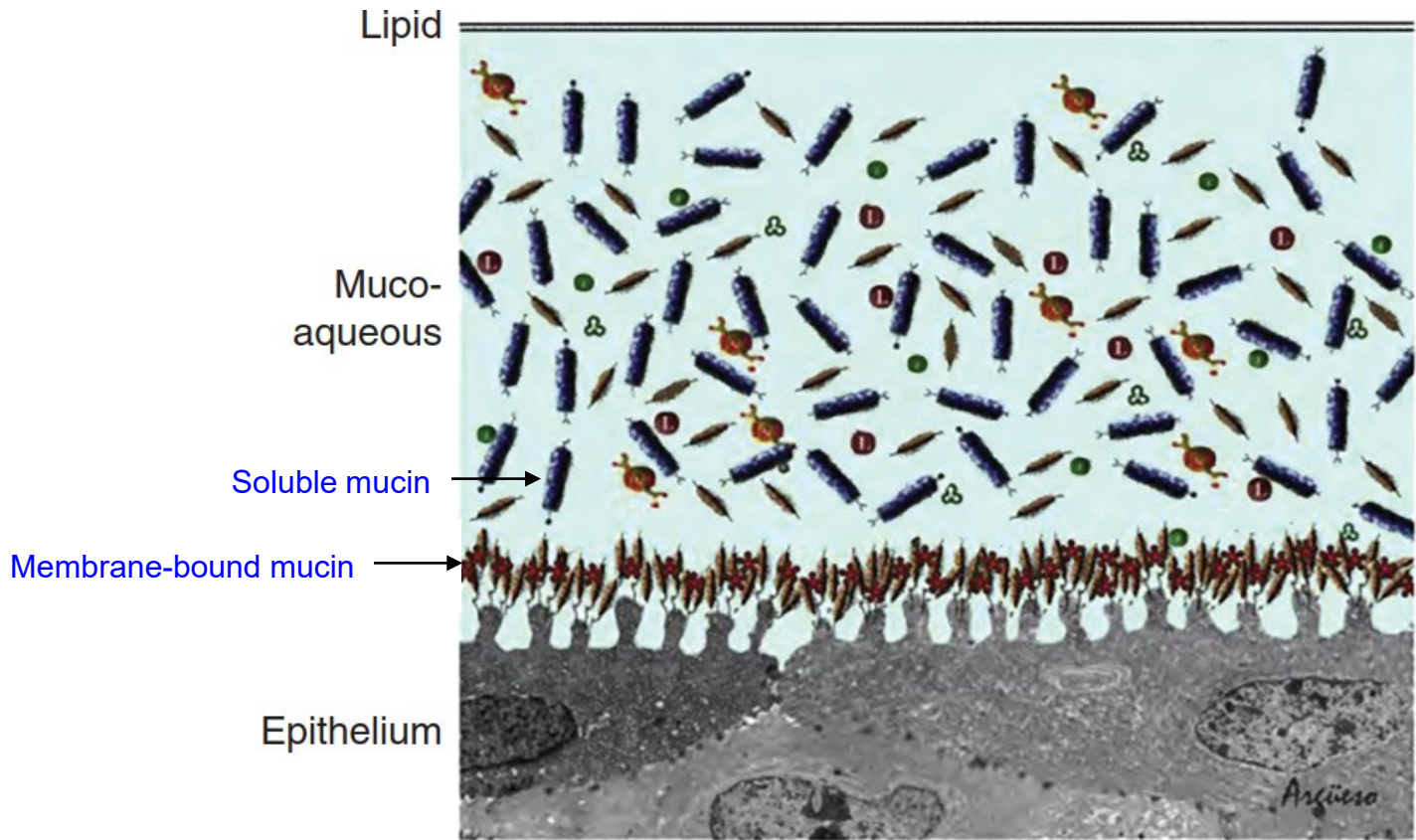
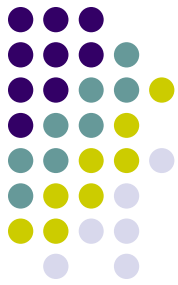
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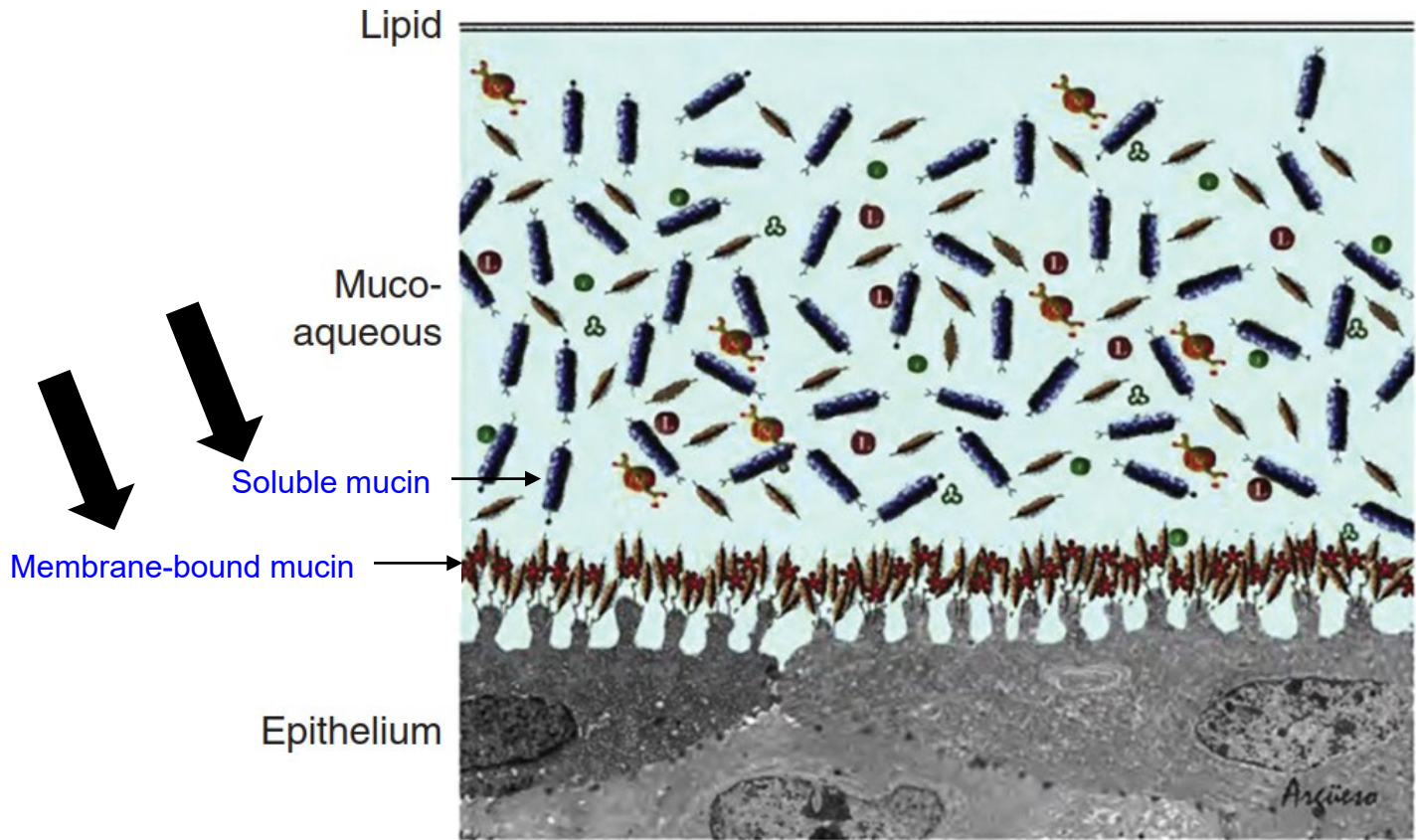
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# Dry Eye Syndrome



We saw this depiction of the *two-phase model of the tear film* earlier in the set... But are now ready to note the presence and location of mucin.

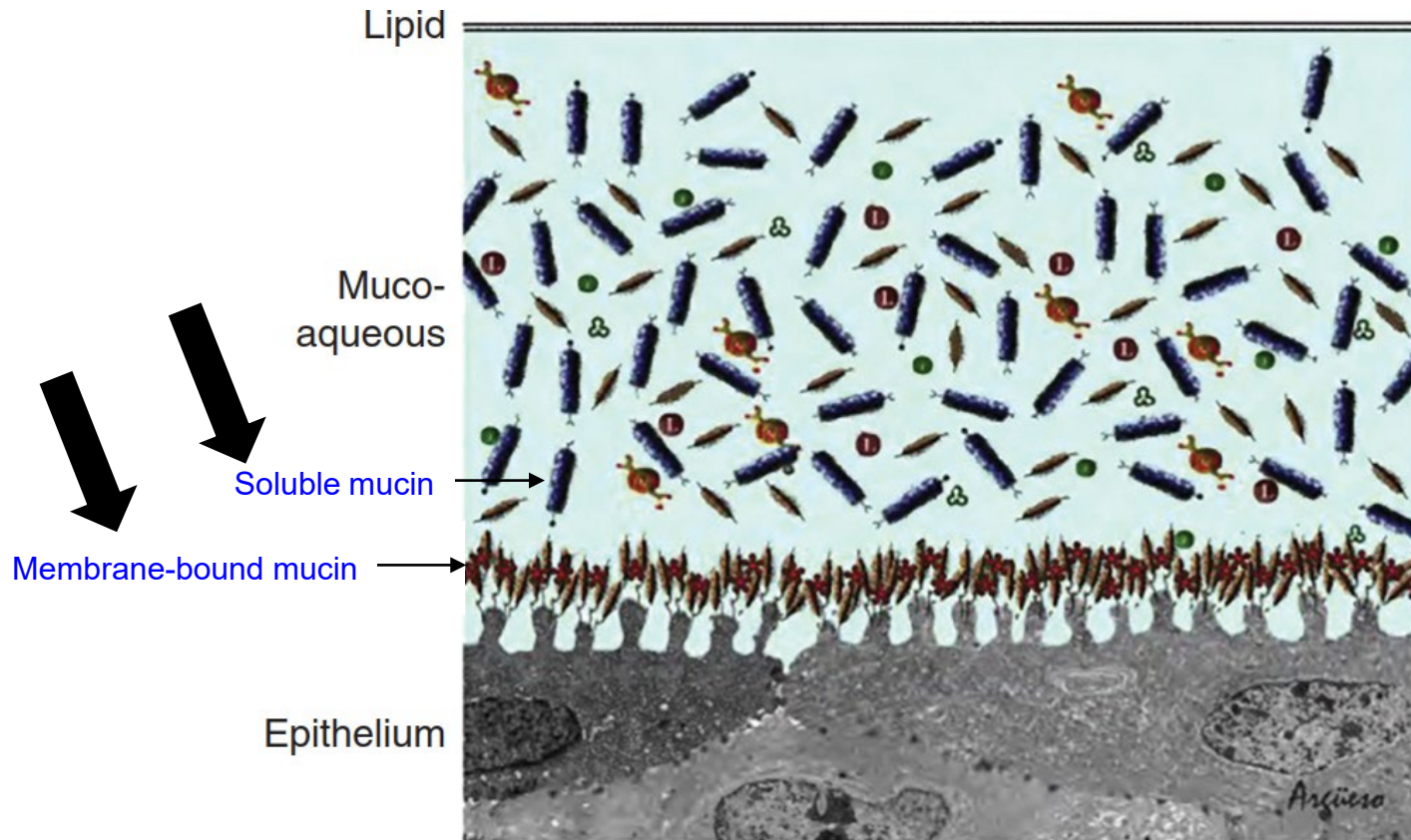
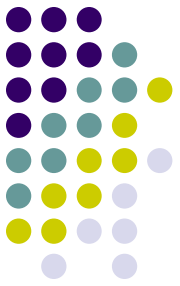
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We saw this depiction of the *two-phase model of the tear film* earlier in the set... But are now ready to note the presence and location of mucin. Note that in addition to the 'soluble' mucins of the mucoaqueous layer, there are 'membrane-bound' mucins contributing to the structure of the corneal epithelium.

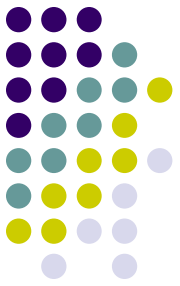


# Dry Eye Syndrome

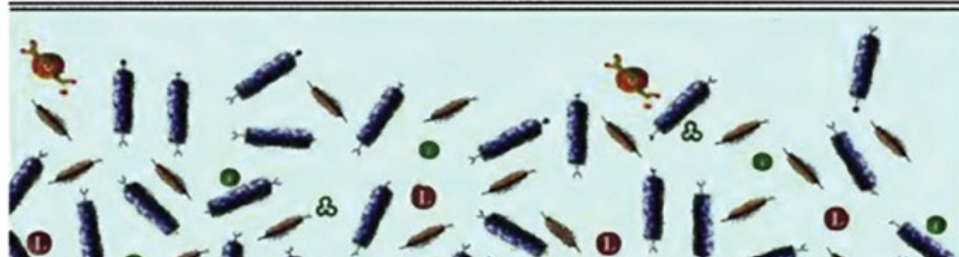


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# Dry Eye Syndrome



Lipid

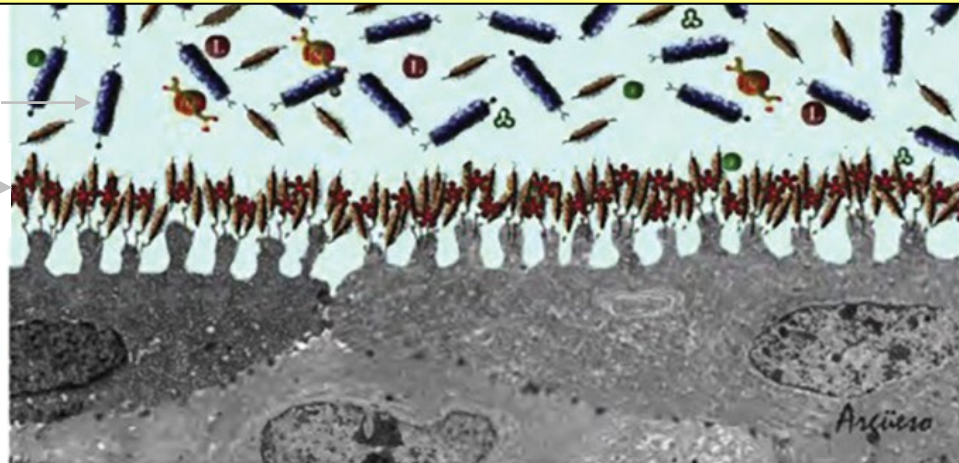


*For more on the tear film, see slide-set K47*

Soluble mucin

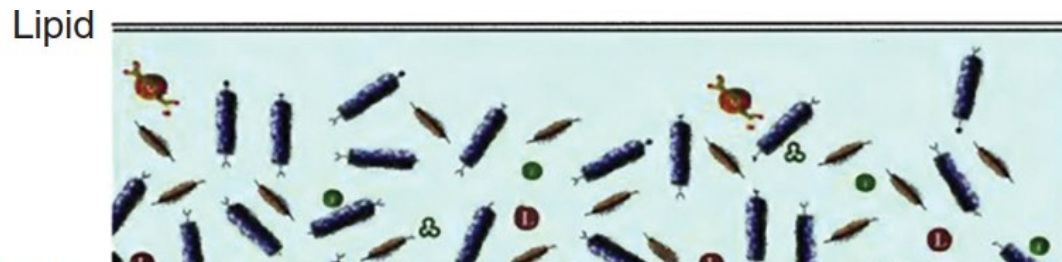
Membrane-bound mucin

Epithelium

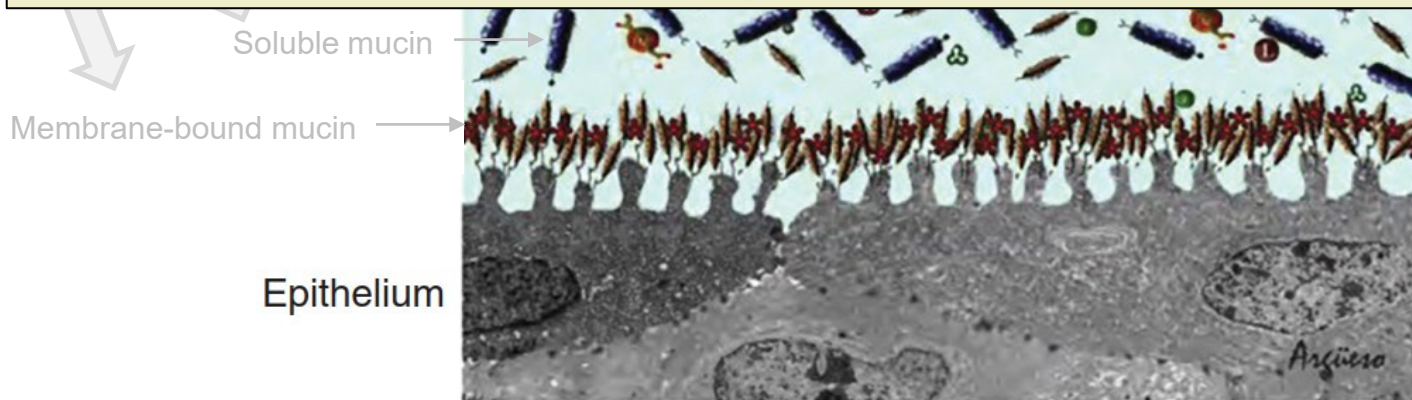


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# Dry Eye Syndrome



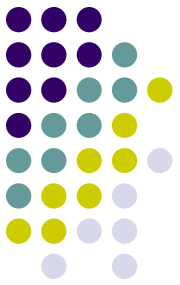
Next we will look at the *Lacrimal Functional Unit (LFU)* and its role in tear production and maintenance



We saw this depiction of the *two-phase model of the tear film* earlier in the set... But are now ready to note the presence and location of mucin. Note that in addition to the 'soluble' mucins of the mucoaqueous layer, there are 'membrane-bound' mucins contributing to the *glycocalyx* of the corneal epithelium.

# Dry Eye Syndrome

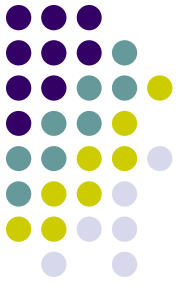
## *The LFU*



*What is the lacrimal functional unit (LFU)?*

# Dry Eye Syndrome

## *The LFU*



*What is the* lacrimal functional unit (*LFU*)?

The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film

# Dry Eye Syndrome

## *The LFU*

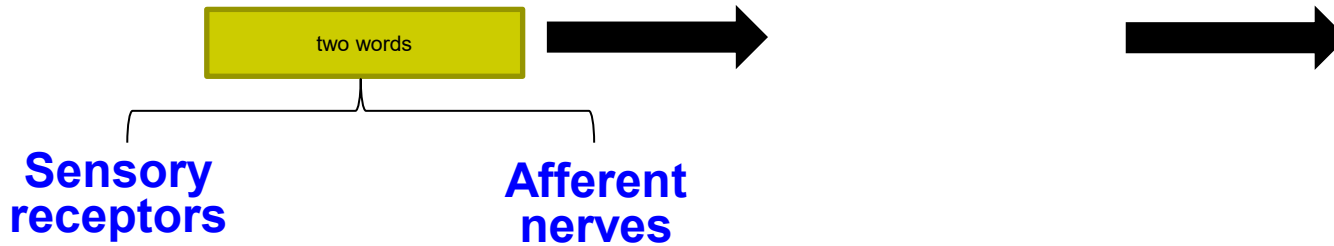
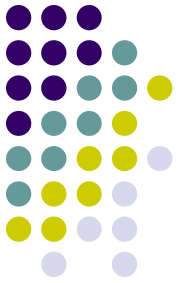


*What is the lacrimal functional unit (LFU)?*

The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it as the reflex arc responsible for the production of the components of the tear film.

# Dry Eye Syndrome

## *The LFU*



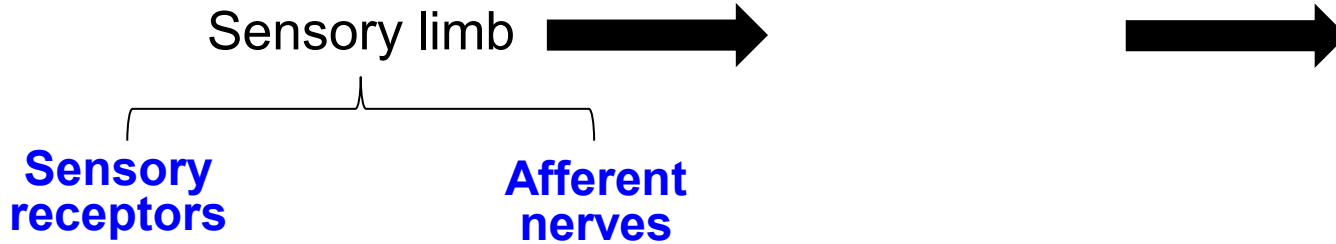
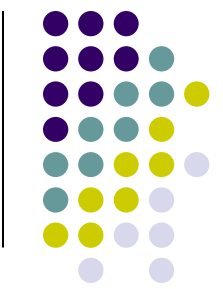
Recall that a reflex arc has three components: A two words consisting of sensory receptors and afferent nerves

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# Dry Eye Syndrome

## *The LFU*



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# Dry Eye Syndrome

## The LFU

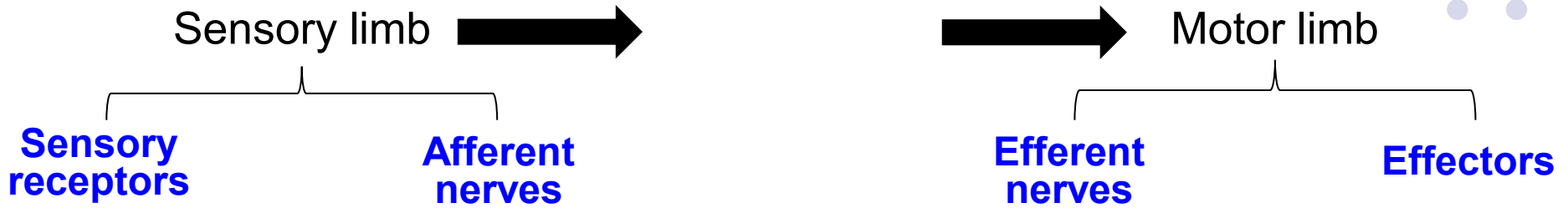
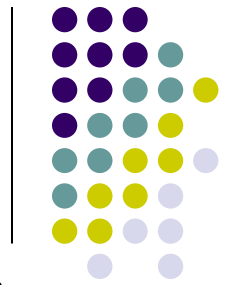


Recall that a reflex arc has three components: A *sensory limb* consisting of sensory receptors and afferent nerves, a two words consisting of efferent nerves and the effector end-organ

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# Dry Eye Syndrome

## The LFU

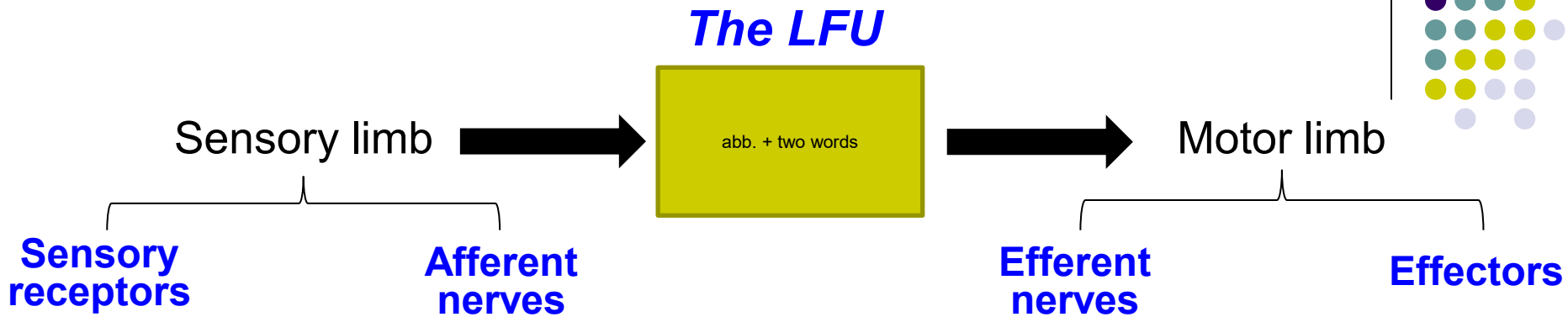


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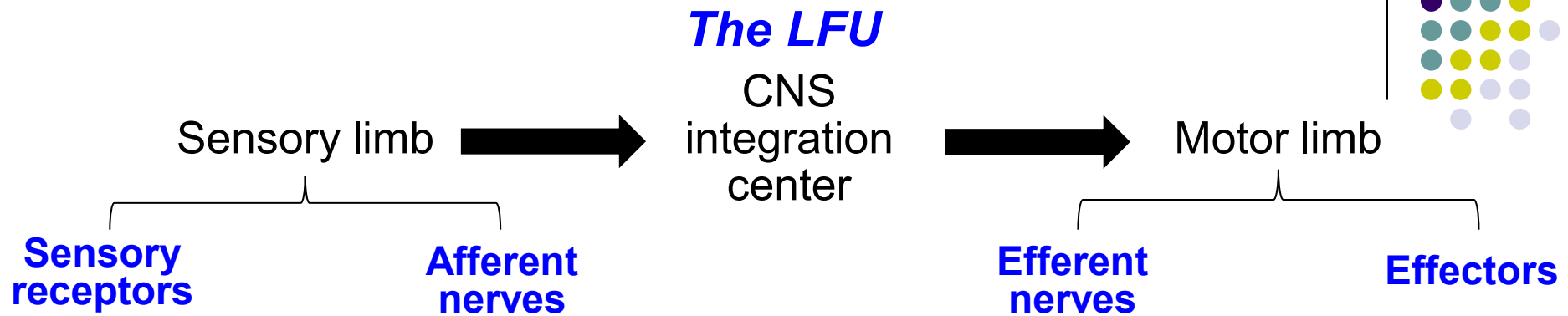


Recall that a reflex arc has three components: A *sensory limb* consisting of sensory receptors and afferent nerves, a *motor limb* consisting of efferent nerves and the effector end-organ, and a **LFU** (abb. + two words) that connects the afferent and efferent limbs.

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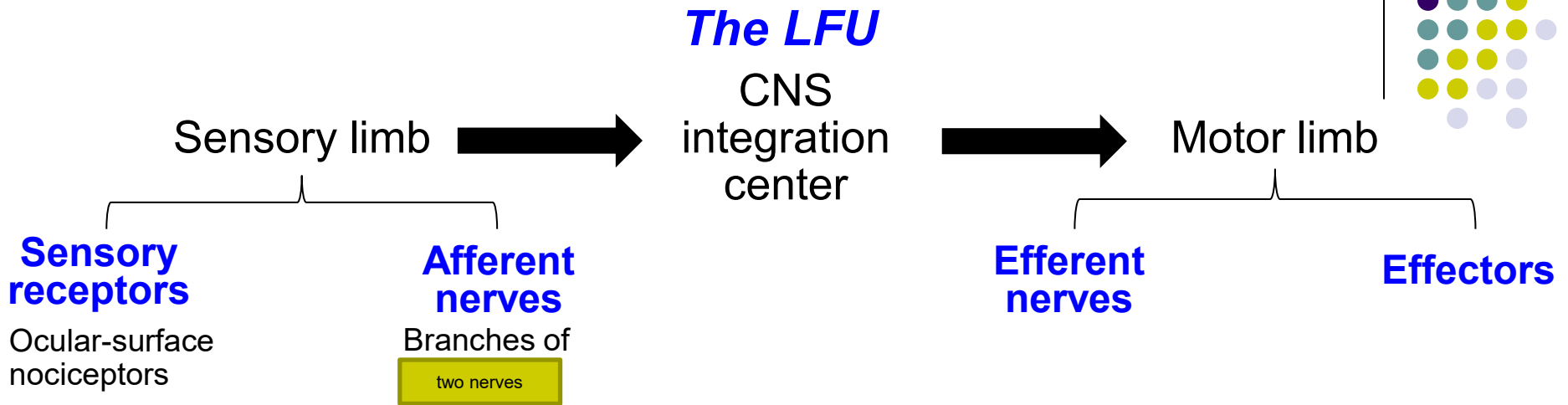
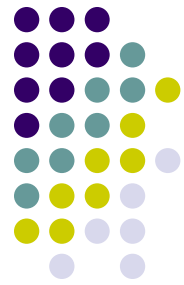


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# Dry Eye Syndrome



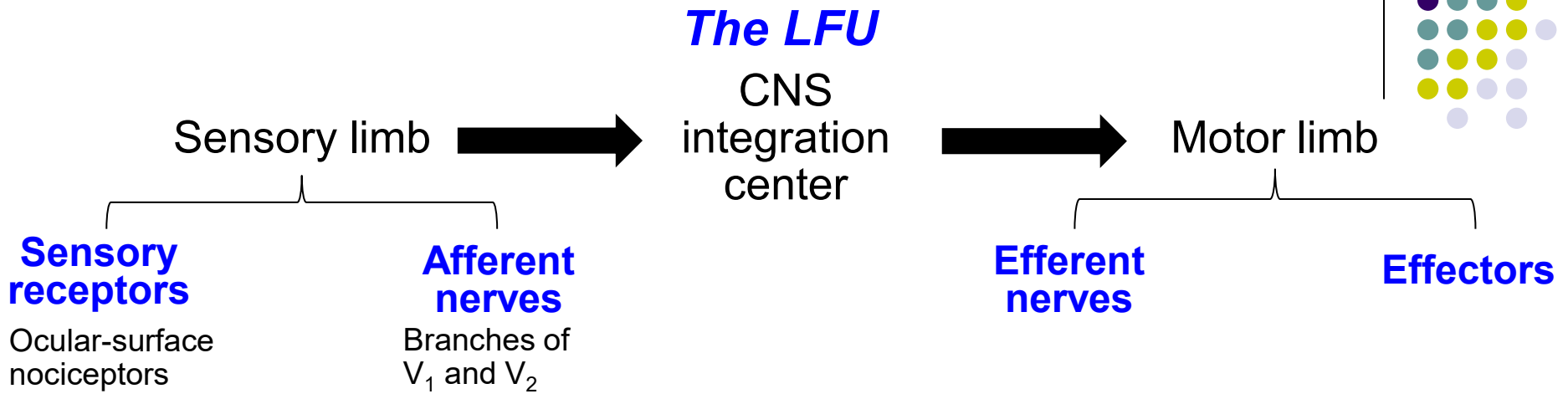
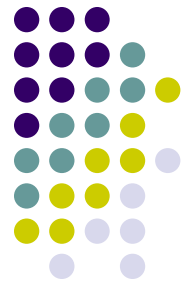
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*What is the lacrimal functional unit (LFU)?*

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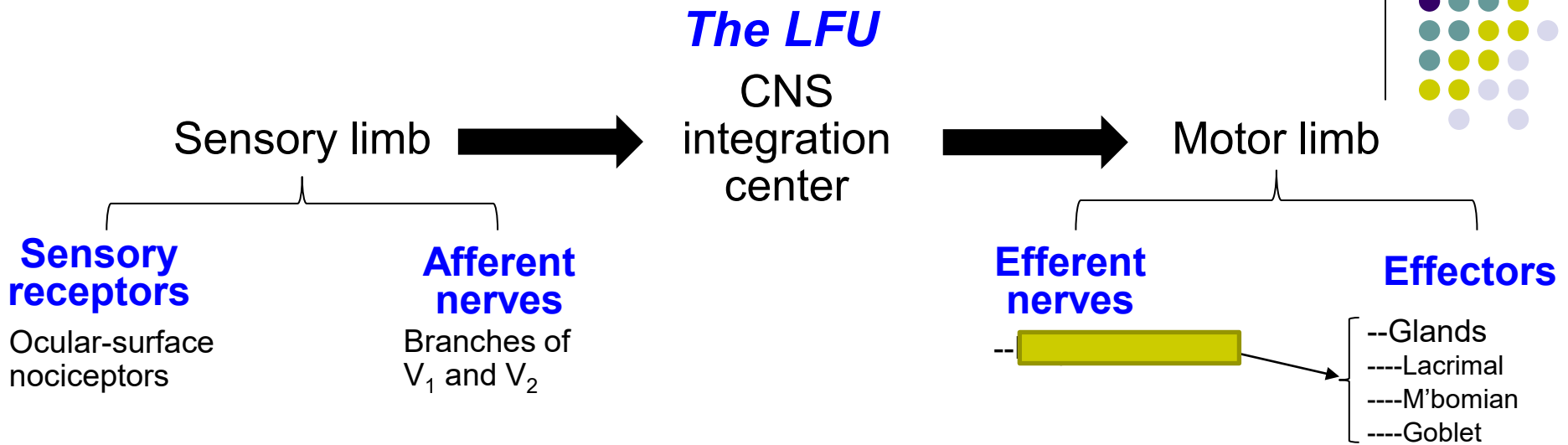
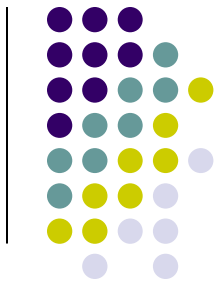


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*What is the lacrimal functional unit (LFU)?*  
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# Dry Eye Syndrome



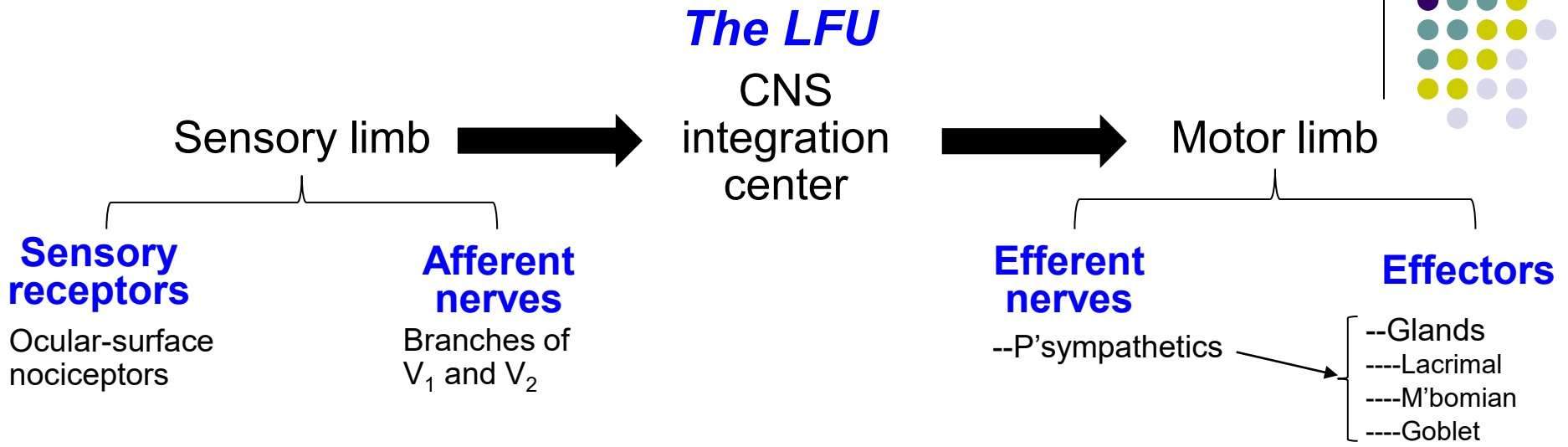
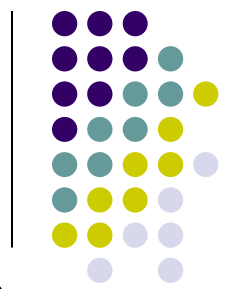
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*What is the lacrimal functional unit (LFU)?*

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# Dry Eye Syndrome



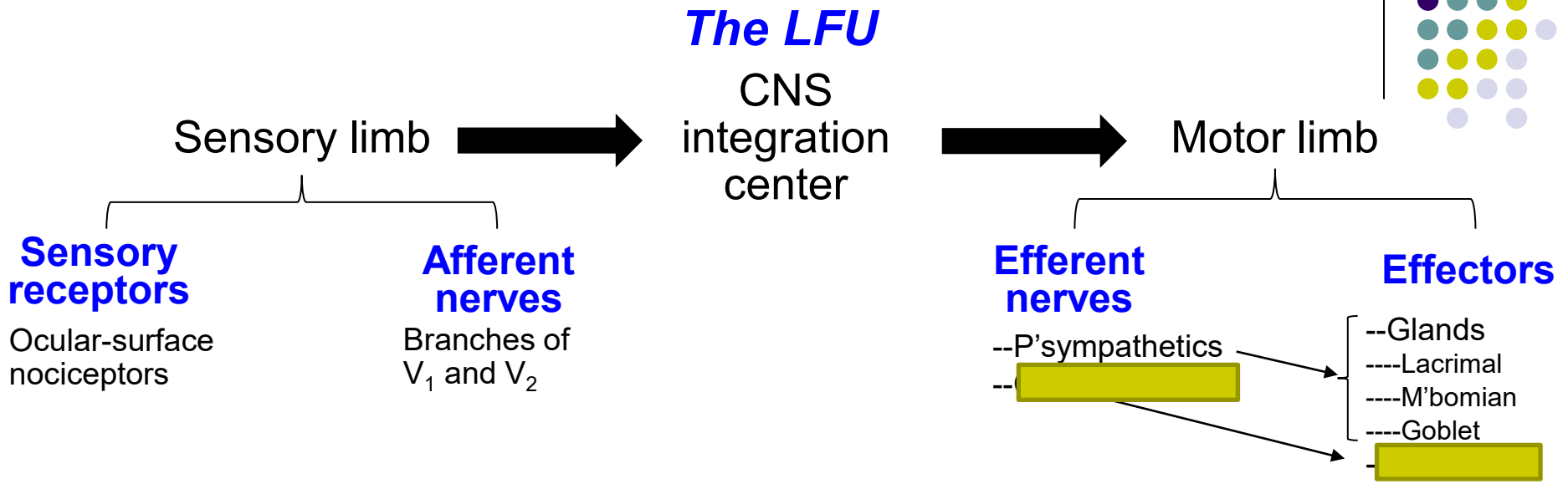
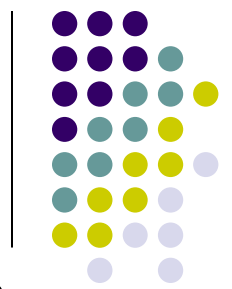
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# Dry Eye Syndrome

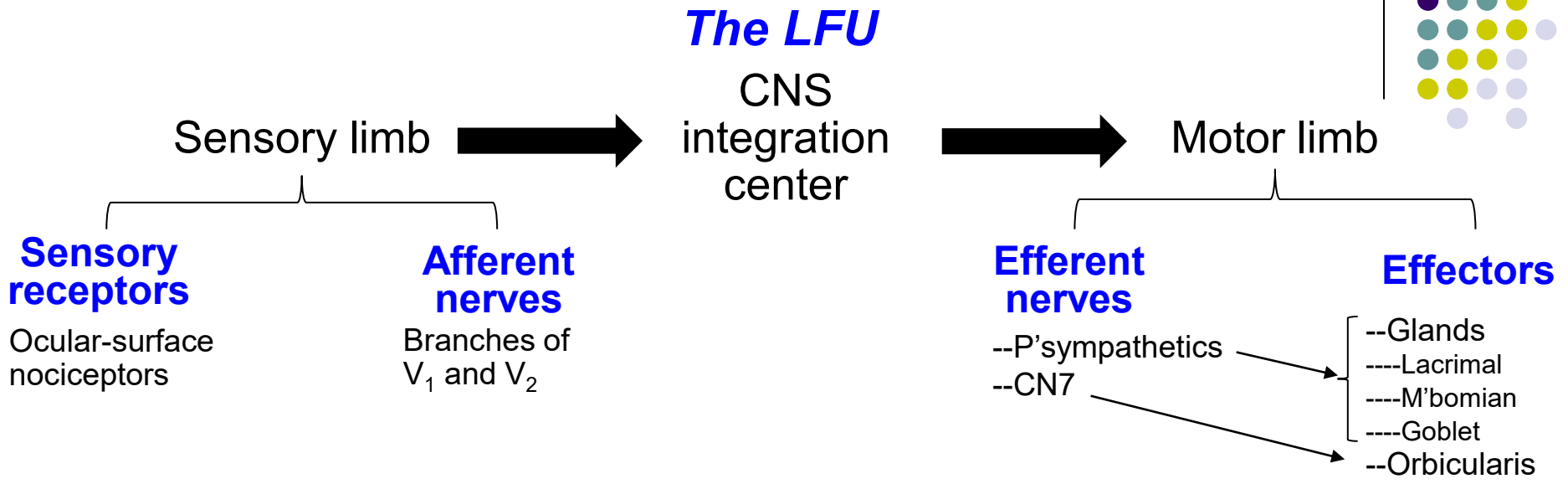
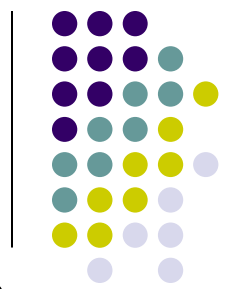


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*What is the lacrimal functional unit (LFU)?*  
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# Dry Eye Syndrome



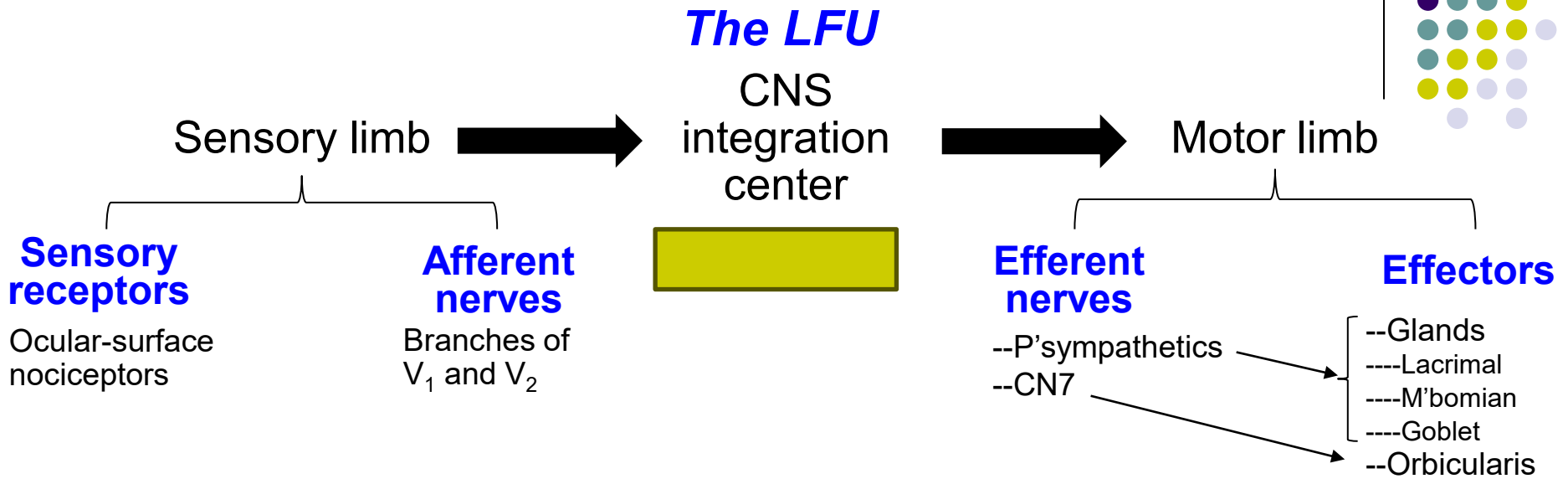
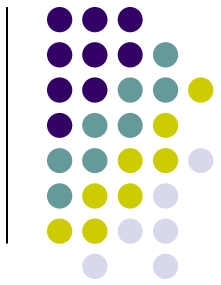
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The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it **the reflex arc responsible for the production of the components of the tear film.**

# Dry Eye Syndrome

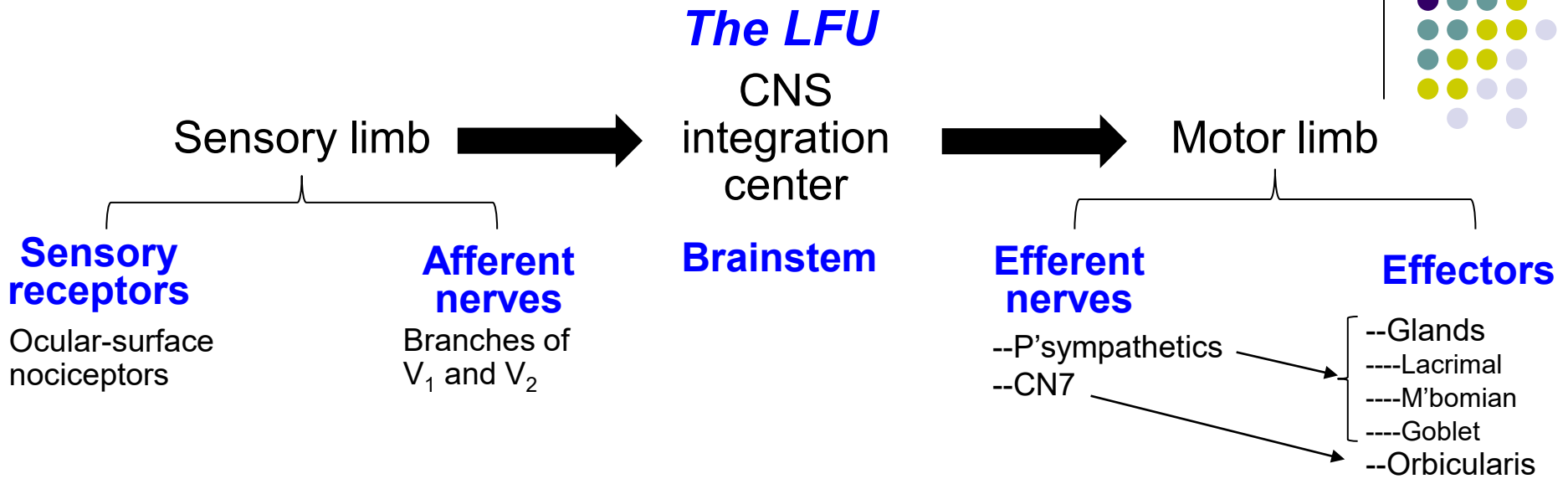
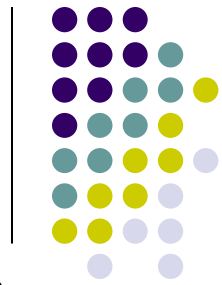


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*What is the lacrimal functional unit (LFU)?*  
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# Dry Eye Syndrome



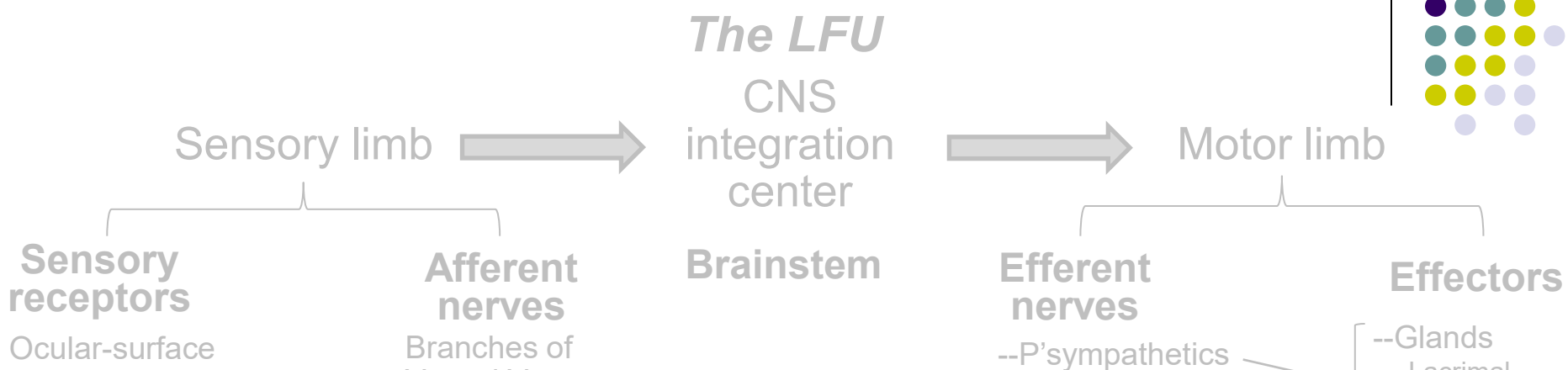
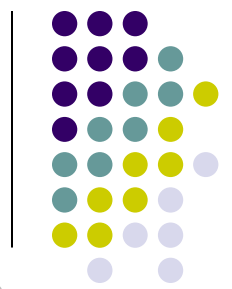
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# Dry Eye Syndrome



**For more on the LFU, see slide-set K46**

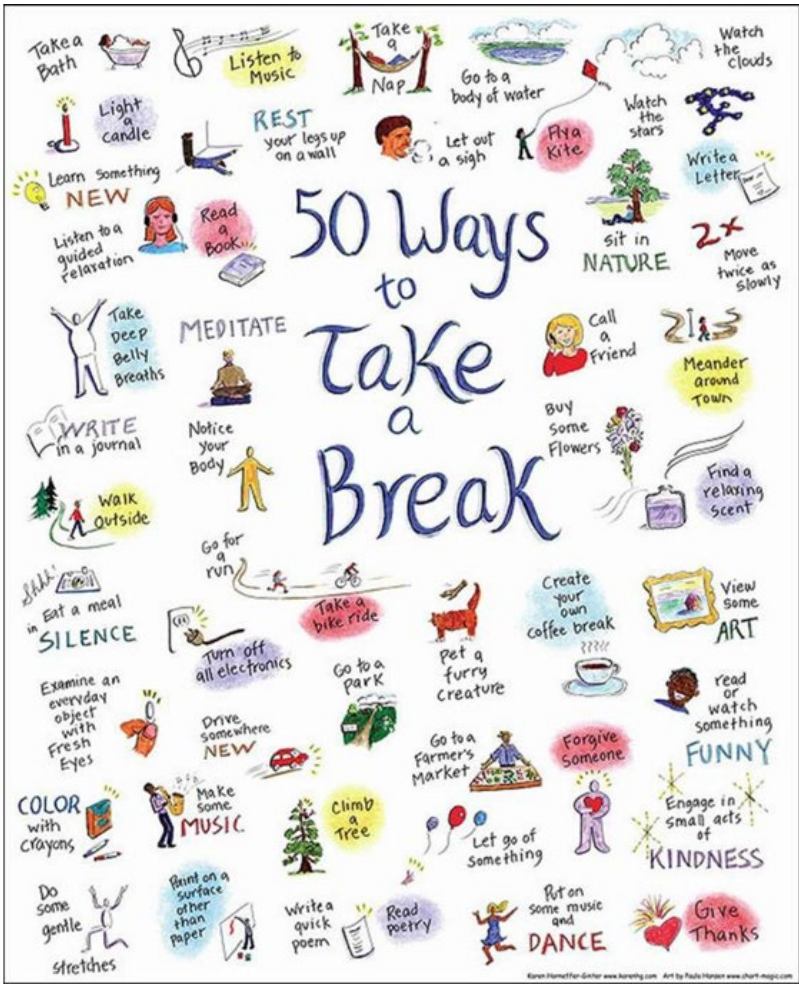
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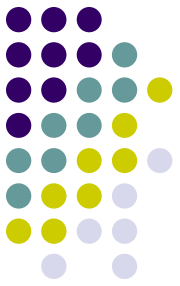
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# Dry Eye Syndrome



(This is a good point in the set to take a break)

# Dry Eye Syndrome



*We are ready (finally!) to tackle the pathophysiology of DES...*

# Dry Eye Syndrome



The pathophysiology for DES damage starts with derangement of the tear film in the form of

**Tear** word

*We are ready (finally!) to tackle the pathophysiology of DES...  
Which commences with something the importance of which  
was stressed earlier in the slide-set*

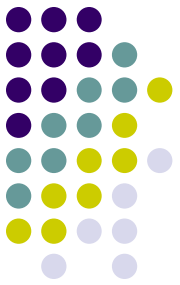
**Tear**

same word



## Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**



*We are ready (finally!) to tackle the pathophysiology of DES...  
Which commences with something the importance of which  
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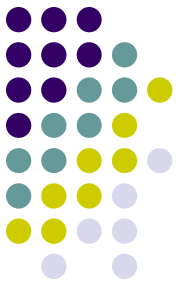
Tear  
hyperosmolarity

## Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

*(Reiterating for emphasis)*

*What are the units of measurement for tear-film osmolarity?*



**Tear  
hyperosmolarity**

## Dry Eye Syndrome

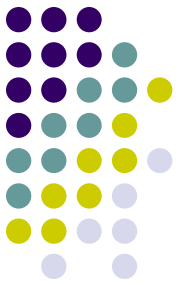
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*(Reiterating for emphasis)*

*What are the units of measurement for tear-film osmolarity?*

*milli-osmols per liter (mOsm/L)*

**Tear  
hyperosmolarity**



## Dry Eye Syndrome

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*(Reiterating for emphasis)*

*What are the units of measurement for tear-film osmolarity?*

milli-osmols per liter (mOsm/L)

*What is the osmolarity of the normal tear film?*

**Tear  
hyperosmolarity**



## Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of  
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*(Reiterating for emphasis)*

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Around 290-300 mOsm/L*

**Tear  
hyperosmolarity**



## Dry Eye Syndrome

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Around 290-300 mOsm/L

*How high does tear osmolarity have to get to be clinically significant?*

**Tear  
hyperosmolarity**

## Dry Eye Syndrome

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Around 290-300 mOsm/L

*How high does tear osmolarity have to get to be clinically significant?*

308 (per EyeWiki)

**Tear  
hyperosmolarity**

## Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**



*In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?*

1) ?

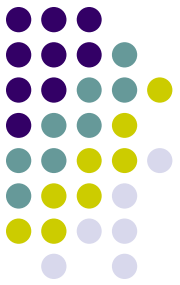
2) ?

*or...*

Tear  
hyperosmolarity



## Dry Eye Syndrome



The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

*In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?*

1) The amount of aqueous  can be inadequate to maintain normal osmolarity.

*or...*

2) The amount of aqueous  can be too high to maintain normal osmolarity.

Tear  
hyperosmolarity

## Dry Eye Syndrome



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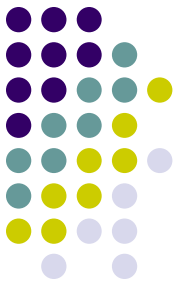
1) The amount of aqueous produced can be inadequate to maintain normal osmolarity.

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Tear  
hyperosmolarity

## Dry Eye Syndrome



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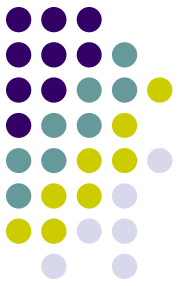
**This state is known as...**

2) The amount of aqueous lost can be too high to maintain normal osmolarity.

?

→ Tear  
hyperosmolarity

## Dry Eye Syndrome



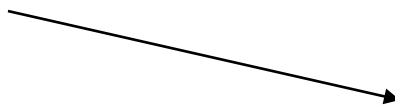
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1) The amount of aqueous produced can be inadequate to maintain normal osmolarity.

**This state is known as...**

*Aqueous Tear  
Deficiency*



**Tear  
hyperosmolarity**

2) The amount of aqueous lost can be too high to maintain normal osmolarity.

## Dry Eye Syndrome



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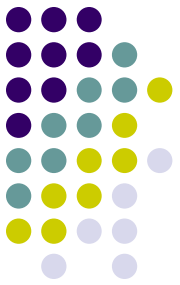
**This state is known as...**

?

Tear  
hyperosmolarity

A diagram showing two arrows pointing towards the central text 'Tear hyperosmolarity'. The arrow from the left originates from the text 'Aqueous Tear Deficiency' and points to the 'Tear' part of the central text. The arrow from the right originates from a question mark '?' and points to the 'Tear' part of the central text.

## Dry Eye Syndrome



The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

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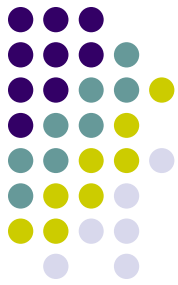
2) The amount of aqueous lost can be too high to maintain normal osmolarity.

**This state is known as...**

*Evaporative  
Dry Eye*

→ Tear  
hyperosmolarity ←

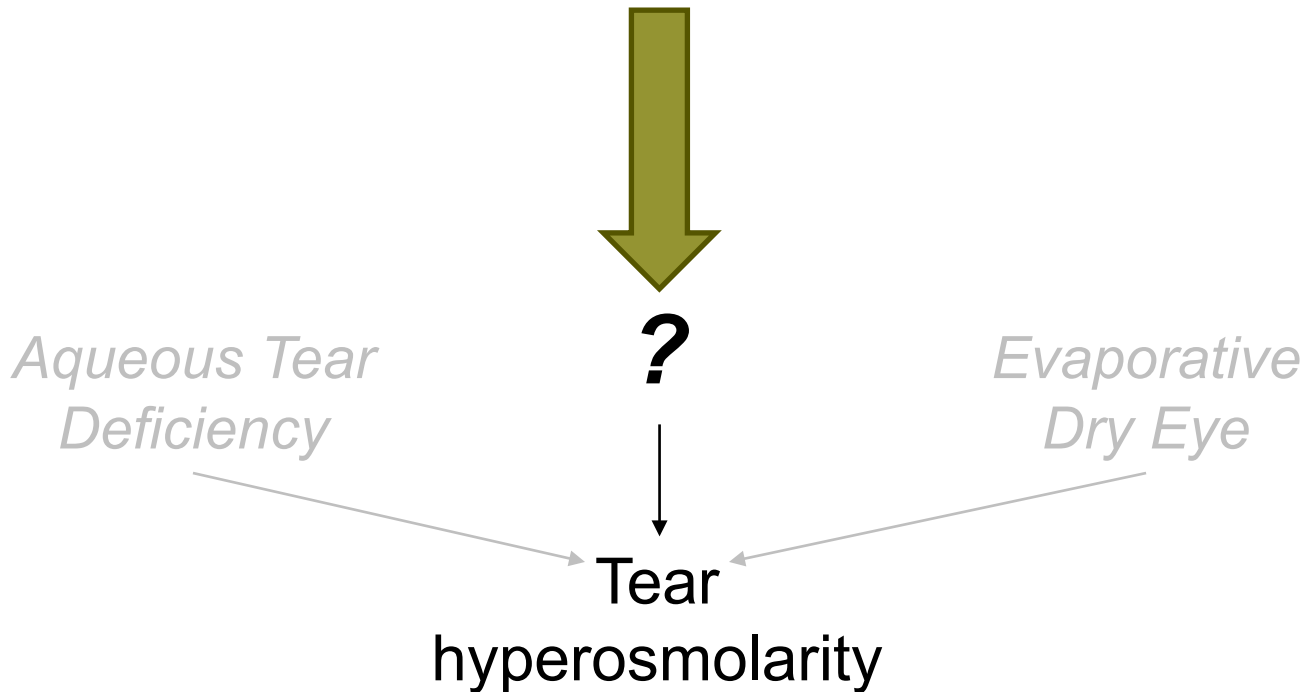
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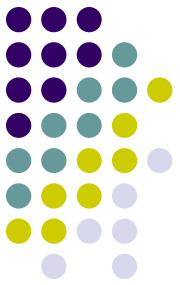
The pathophysiology for DES damage starts with

**Head's up:** Later in the set we're gonna add a *third* mechanism leading to tear hyperosmolarity

*three*  
In what ~~two~~ fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

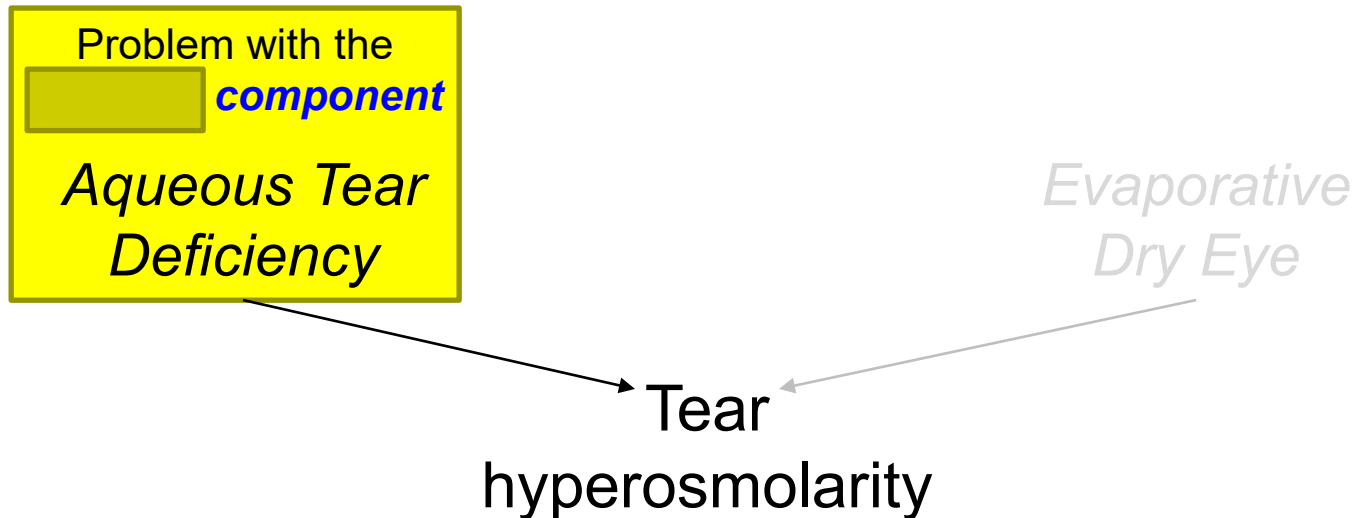


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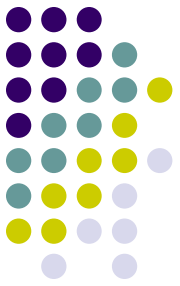
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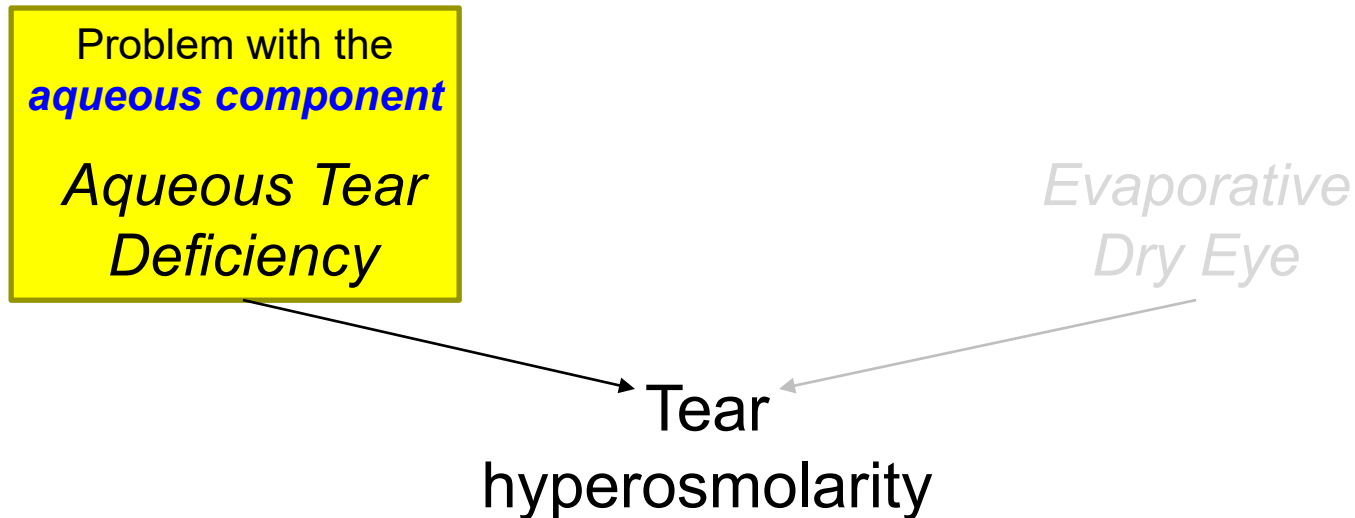


## Dry Eye Syndrome



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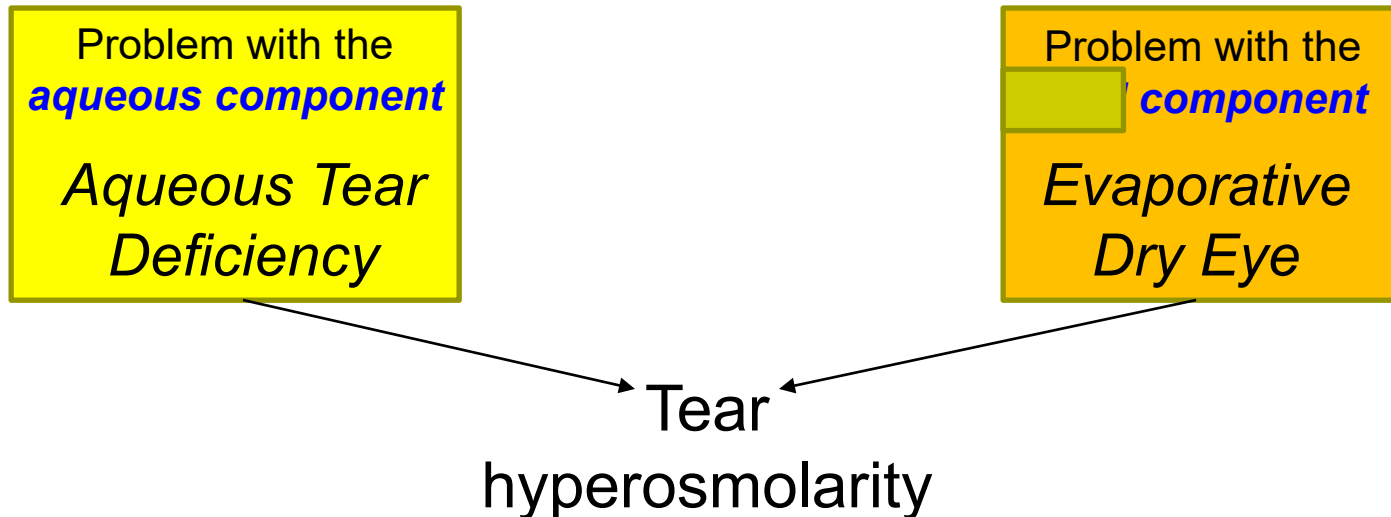


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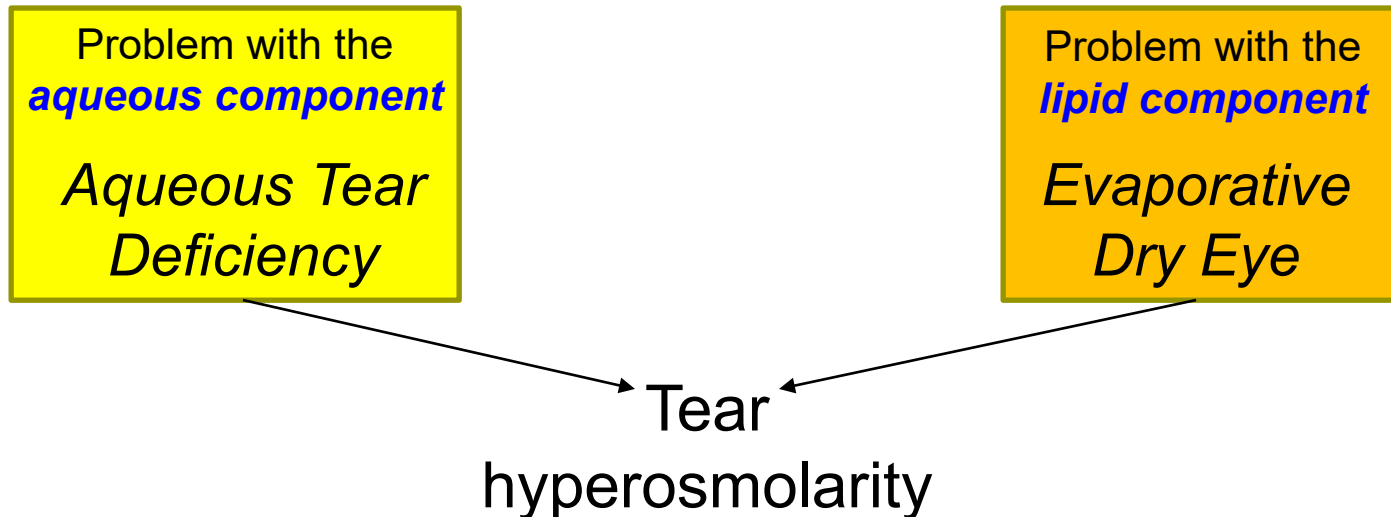


## Dry Eye Syndrome



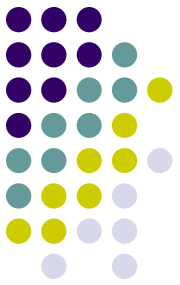
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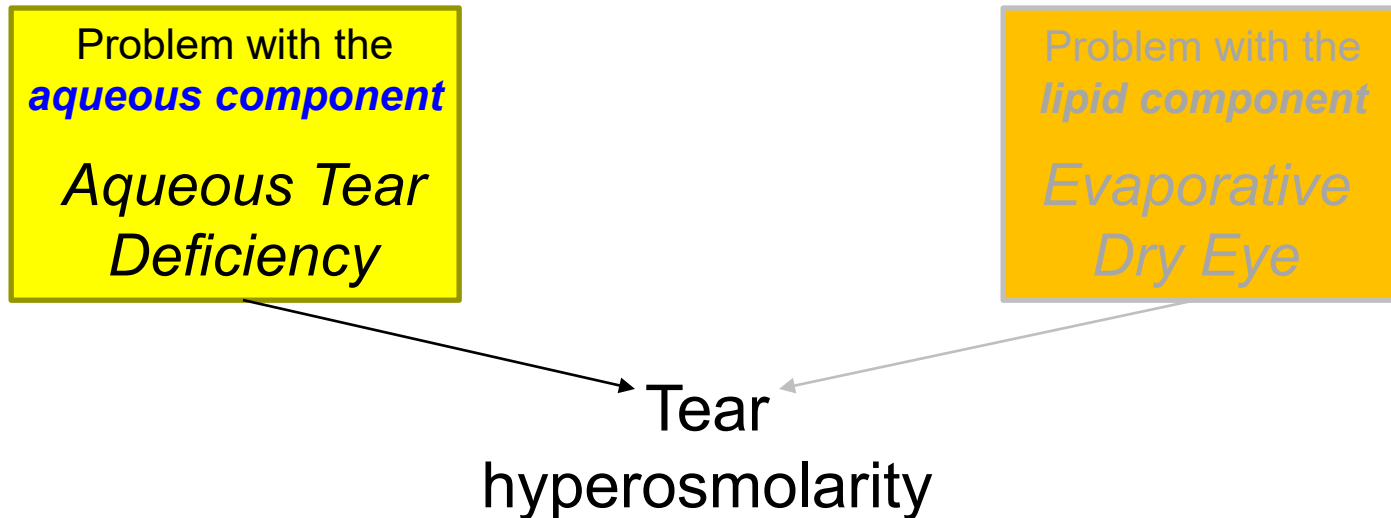


## Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.



Let's drill down on both, starting with ATD.



# Dry Eye Syndrome

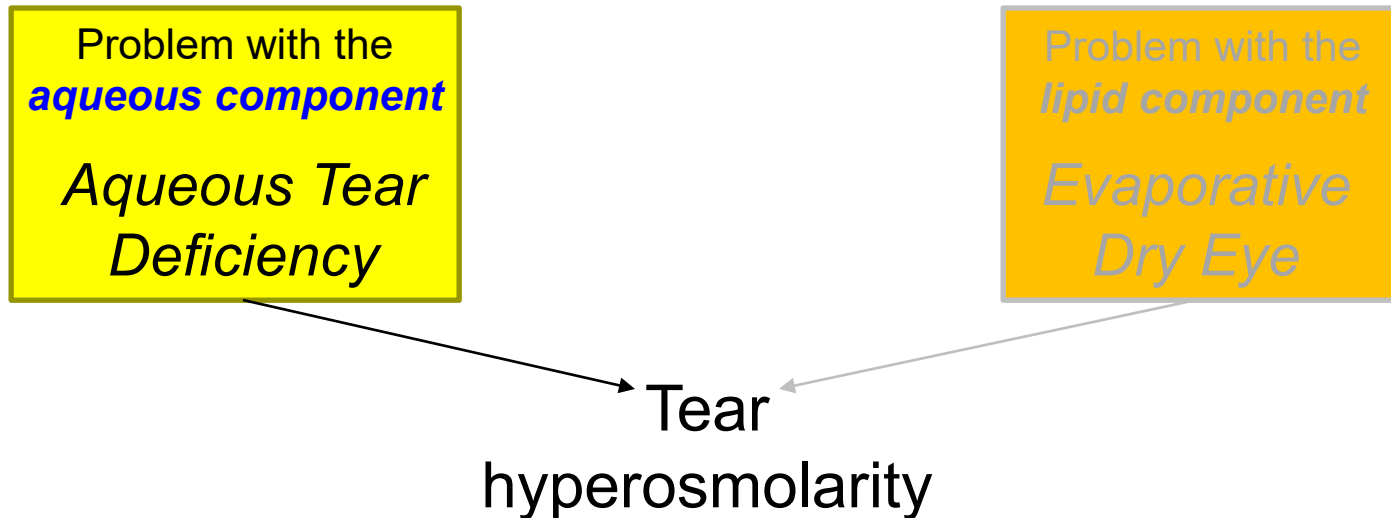


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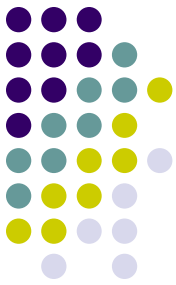
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*Wait for it...*

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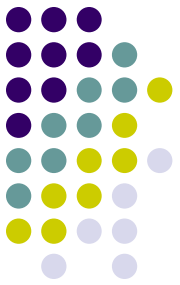


*What are the three classic tests of aqueous tear production?*

*Wait for it...OK, now answer*

Test name			
?			
?			
?			

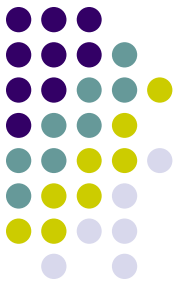
# Dry Eye Syndrome



*What are the three classic tests of aqueous tear production?*

Test name			
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<i>Schirmer I</i>			
<i>Schirmer II</i>			

# Dry Eye Syndrome



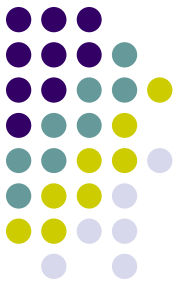
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# Dry Eye Syndrome



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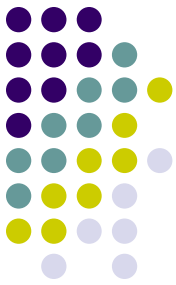


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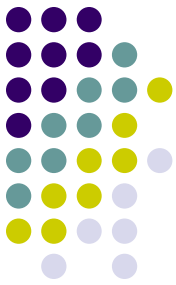


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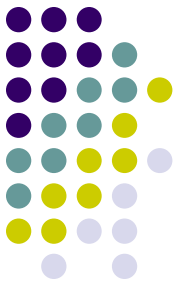


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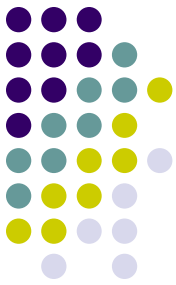


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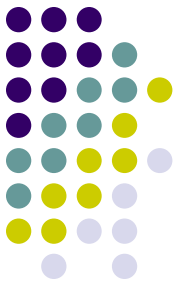


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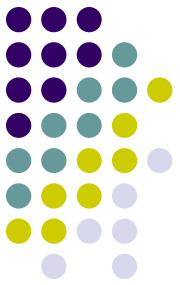


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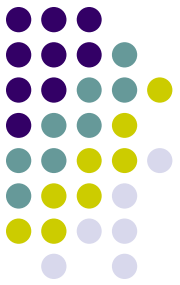
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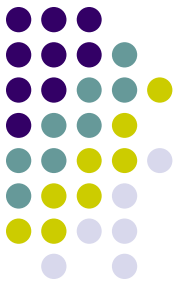


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# Dry Eye Syndrome



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# Dry Eye Syndrome



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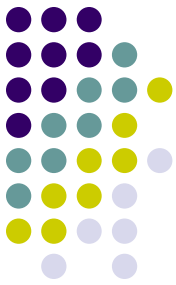


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Test name	Assesses...	Protocol	Interpretation
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# Dry Eye Syndrome

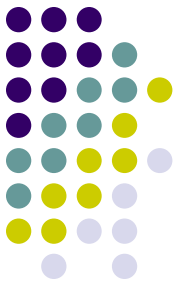


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Test name	Assesses...	Protocol	Interpretation
<i>Basal secretion test</i>	Basal secretion (duh)	Instill anesthetic, blot, place strip, measure saturation at 5 min	Less than 3 mm wetting after 5 min = ATD
<i>Schirmer I</i>	Basal and reflex secretion	Same, but <b>without</b> instilling anesthetic	
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# Dry Eye Syndrome

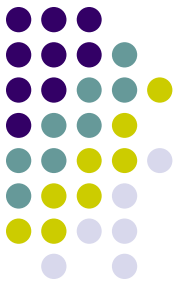


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# Dry Eye Syndrome



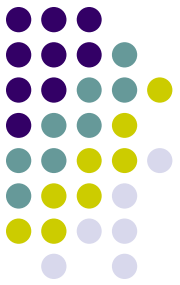
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# Dry Eye Syndrome



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# Dry Eye Syndrome

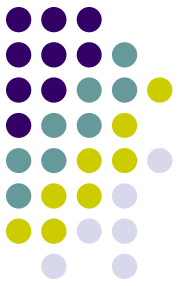


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<i>Schirmer II</i>	Reflex secretion only	Instill anesthetic, blot, place strip, irritate nasal mucosa w/ a cotton-tip	Less than $\frac{\text{amount}}{\text{elapse time}}$ = reflex secretion defect

# Dry Eye Syndrome



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Test name	Assesses...	Protocol	Interpretation
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# Dry Eye Syndrome



What are the...  
What does... (No question—summary slide) ...h interpreted?

Test name	Assesses...	Protocol	Interpretation
<i>Basal secretion test</i>	Basal secretion (duh)	Instill anesthetic, blot, place strip, measure saturation at 5 min	Less than 3 mm wetting after 5 min = ATD
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# Dry Eye Syndrome



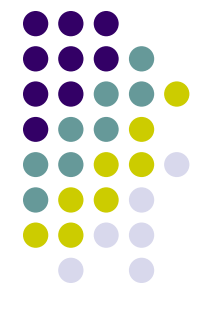
*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

Test name	What are the dimensions of the test strips used?	Interpretation
Basal secretion test		3 mm or 5 min = 0
Schirmer I		5 mm or 5 min = 0
Schirmer II	Reflex secretion only	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome



*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

Test name	What are the dimensions of the test strips used? 30 x 5 mm	Interpretation
Basal secretion test		Less than 3 mm wetting after 5 min =
Schirmer I		Less than 5 mm wetting after 5 min =
Schirmer II	Reflex secretion only	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome



*What are the three classic tests of aqueous tear production?*

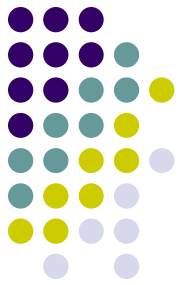
*What does each assess? How is each performed? How is each interpreted?*

Test name	What are the dimensions of the test strips used? 30 x 5 mm	Interpretation
Basal secretion test	How are the strips placed?	Less than 3 mm wetting after 5 min =
Schirmer I		Less than 5 mm wetting after 5 min =
Schirmer II	Reflex secretion only	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*



# Dry Eye Syndrome



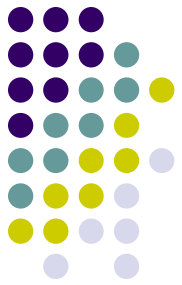
*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

Test name	What are the dimensions of the test strips used?	Interpretation
Basal secretion test	How are the strips placed? With <input type="text" value="length"/> hooked over the lid margin and the other <input type="text" value="length"/> hanging over the front of the lid	3 mm or 5 min =
Schirmer I		5 mm or 5 min =
Schirmer II	Reflex secretion only	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome



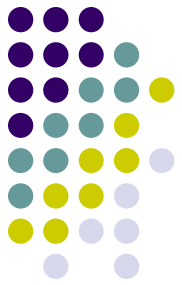
What are the three classic tests of aqueous tear production?

What does each assess? How is each performed? How is each interpreted?

Test name	What are the dimensions of the test strips used?	Interpretation
Basal secretion test	How are the strips placed? With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid	3 mm or 5 mm = 0
Schirmer I		5 mm or 5 min = 0
Schirmer II	Reflex secretion only Instill anesthetic, blot, place strip, irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

(No question—summary slide for review)

# Dry Eye Syndrome



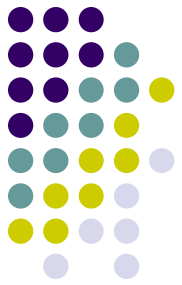
*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

Test name	What are the dimensions of the test strips used?	Interpretation
Basal secretion test	How are the strips placed? With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid	Less than 3 mm wetting after 5 min = defect
Schirmer I	Where along the lid margin should the strip be placed?	Less than 5 mm wetting after 5 min = defect
Schirmer II	Reflex secretion only Place strip, irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome



What are the three classic tests of aqueous tear production?

What does each assess? How is each performed? How is each interpreted?

Test name	What are the dimensions of the test strips used? 30 x 5 mm	Interpretation
Basal secretion test	How are the strips placed? With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid	Less than 3 mm wetting after 5 min = dry eye
Schirmer I	Where along the lid margin should the strip be placed? At the junction of the <input type="text" value="middle"/> proportion and <input type="text" value="outer"/> proportion of the lid	Less than 5 mm wetting after 5 min = dry eye
Schirmer II	Reflex secretion only Place strip, irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

(No question—summary slide for review)

# Dry Eye Syndrome



*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

<b>Test name</b>	<b>What are the dimensions of the test strips used?</b> 30 x 5 mm	<b>Interpretation</b>
<b>Basal secretion test</b>	<b>How are the strips placed?</b> With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid	3 mm or 5 min = 0
<b>Schirmer I</b>	<b>Where along the lid margin should the strip be placed?</b> At the junction of the outer third and middle third of the lid	5 mm or 5 min = 0
<b>Schirmer II</b>	<b>Reflex secretion only</b> Place strip, irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome



*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

Test name	What are the dimensions of the test strips used?	Interpretation
Basal secretion test	How are the strips placed? With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid	3 mm or 5 min = 0
Schirmer I	Where along the lid margin should the strip be placed? At the junction of the outer third and middle third of the lid  We talking upper lid, or lower?	5 mm or 5 min = 0
Schirmer II	Reflex secretion only place strip, irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome



*What are the three classic tests of aqueous tear production?*

*What does each assess? How is each performed? How is each interpreted?*

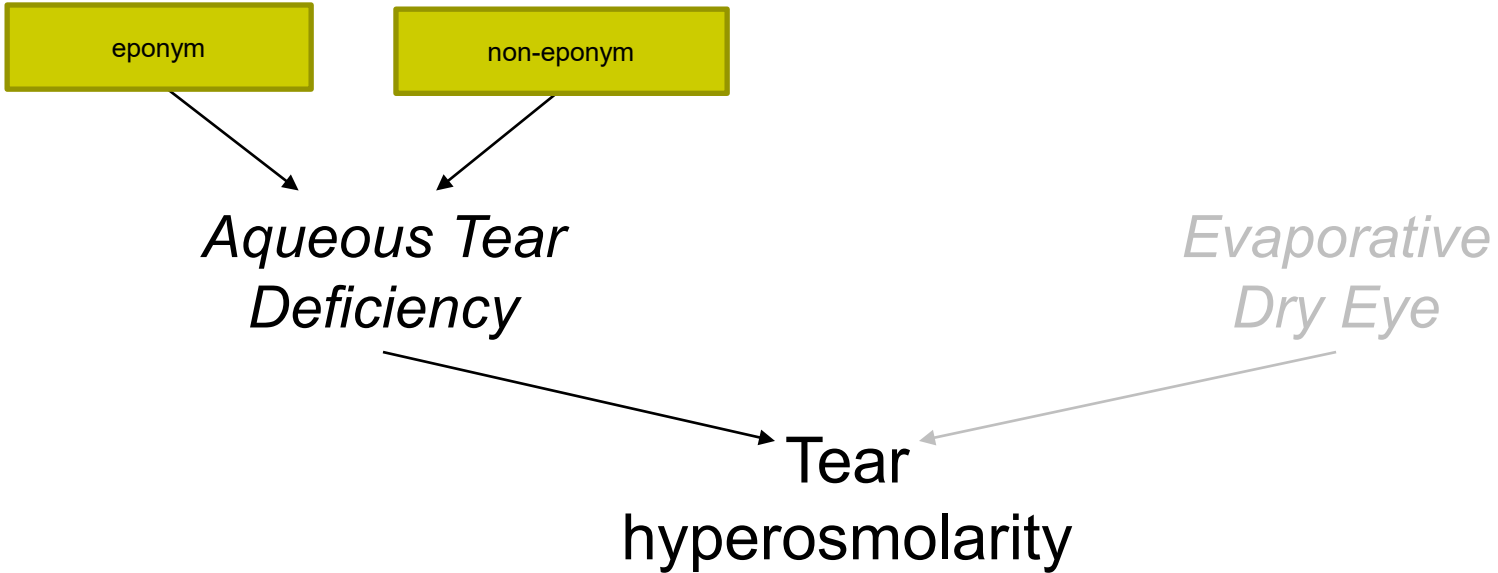
Test name	What are the dimensions of the test strips used?	Interpretation
Basal secretion test	How are the strips placed? With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid	Less than 3 mm wetting after 5 min = defect
Schirmer I	Where along the lid margin should the strip be placed? At the junction of the outer third and middle third of the lid  We talking upper lid, or lower? Seriously?	Less than 5 mm wetting after 5 min = defect
Schirmer II	Reflex secretion only Irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

*(No question—summary slide for review)*

# Dry Eye Syndrome

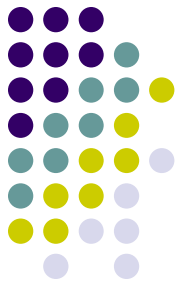


*ATD is subdivided into two categories--what are they?*

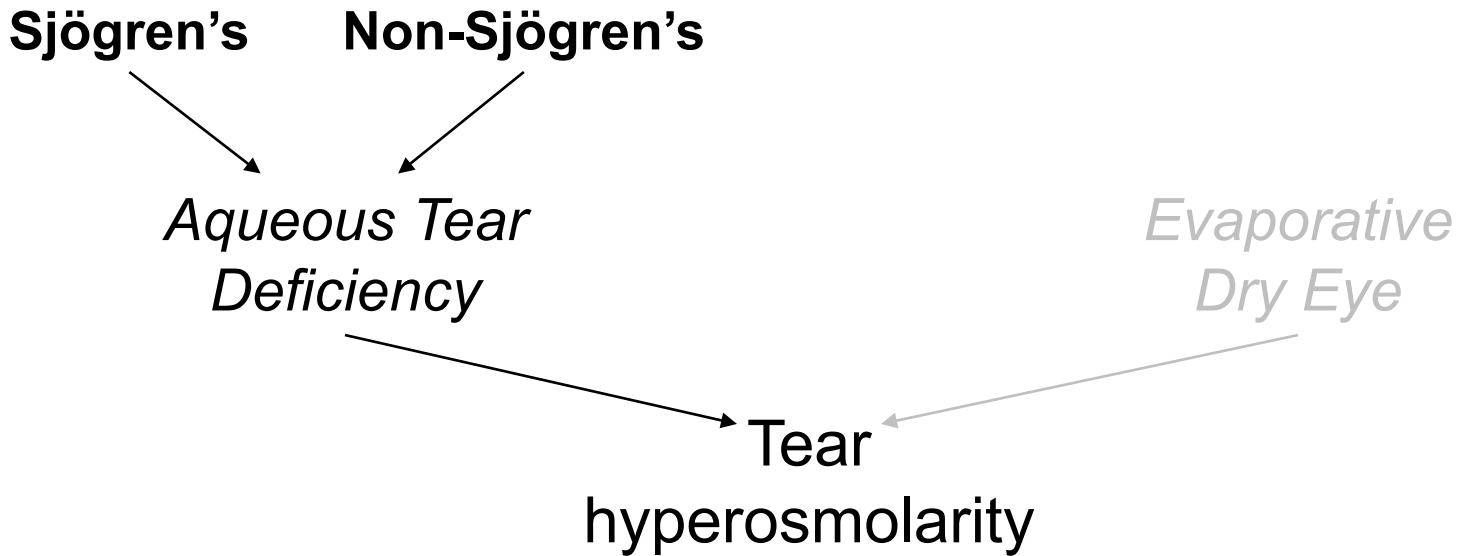




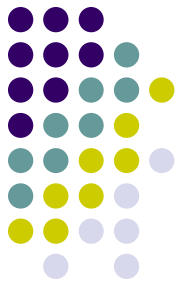
# Dry Eye Syndrome



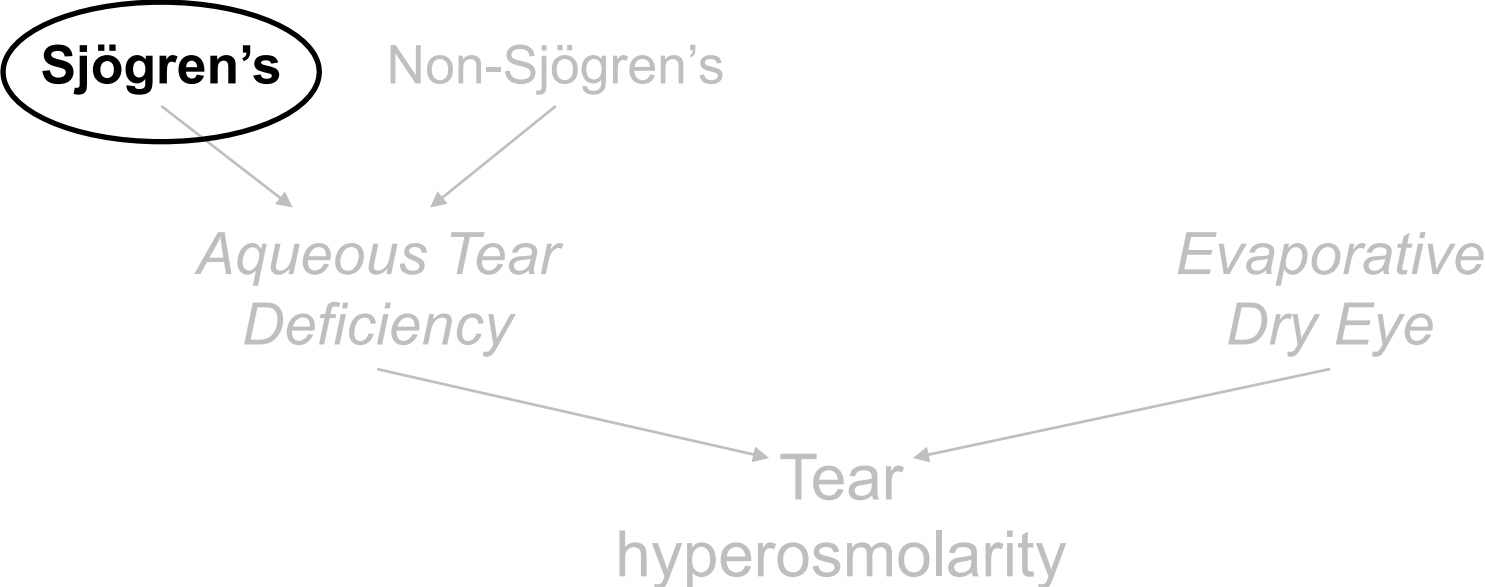
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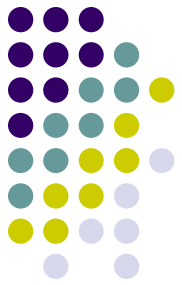
# Dry Eye Syndrome



What is Sjögren's syndrome (SS)?

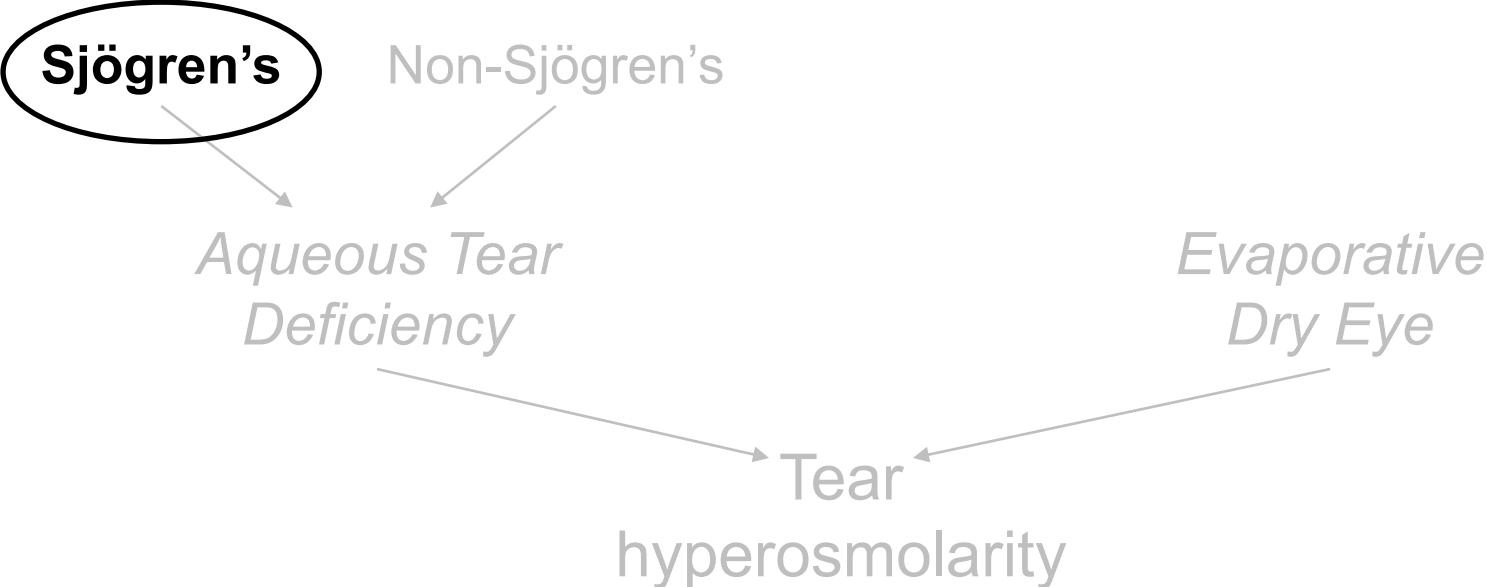


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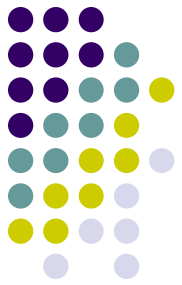


What is Sjögren's syndrome (SS)?

A acute vs chronic autoimmune disorder characterized by cell class infiltration of general type of gland glands

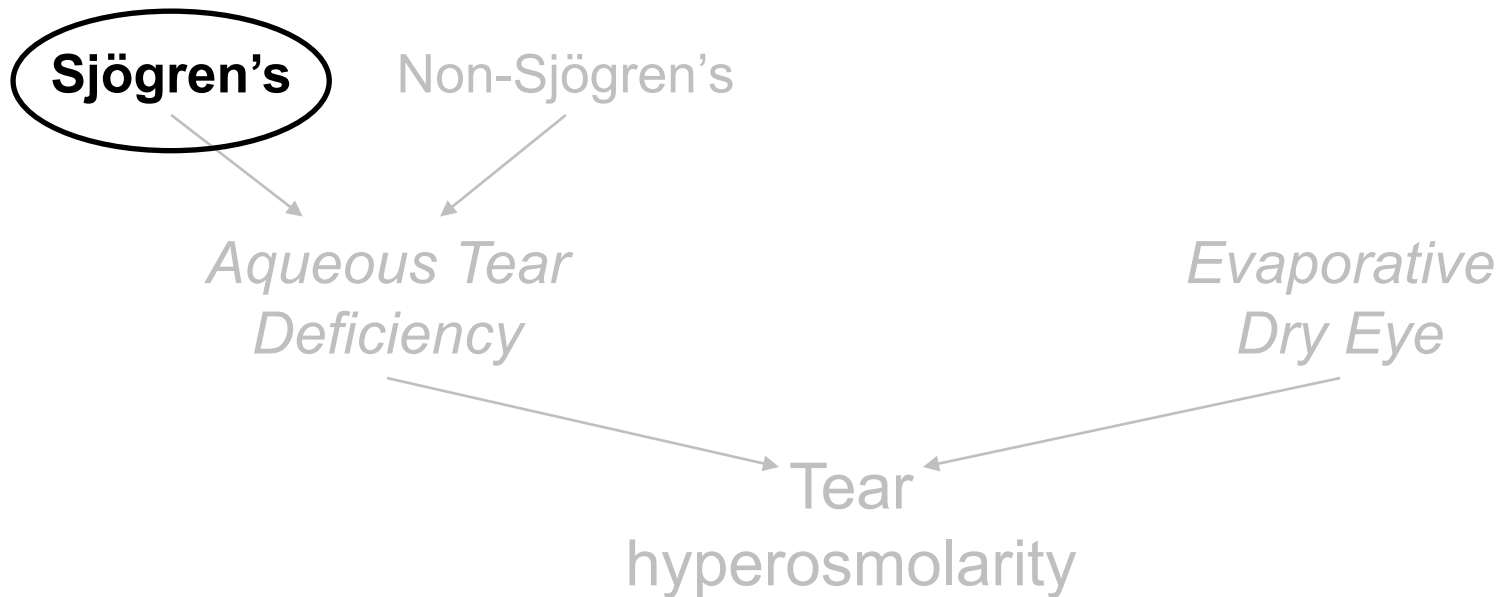


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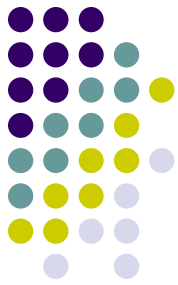


*What is Sjögren's syndrome (SS)?*

A chronic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands



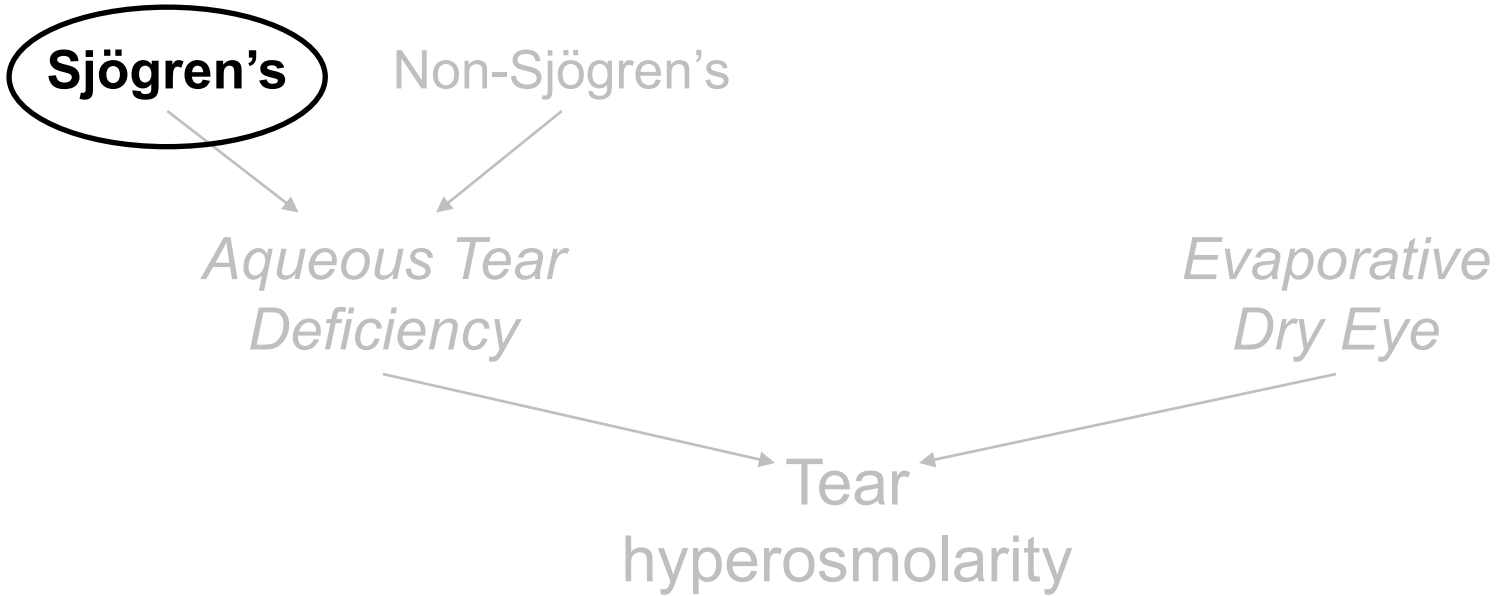
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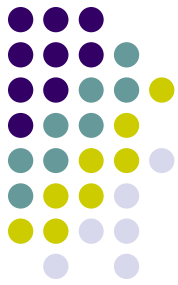
Are the infiltrating lymphocytes T-cells, or B-cells?

What is Sjögren's syndrome (SS)?

A chronic autoimmune disorder characterized by **lymphocytic infiltration** of exocrine glands



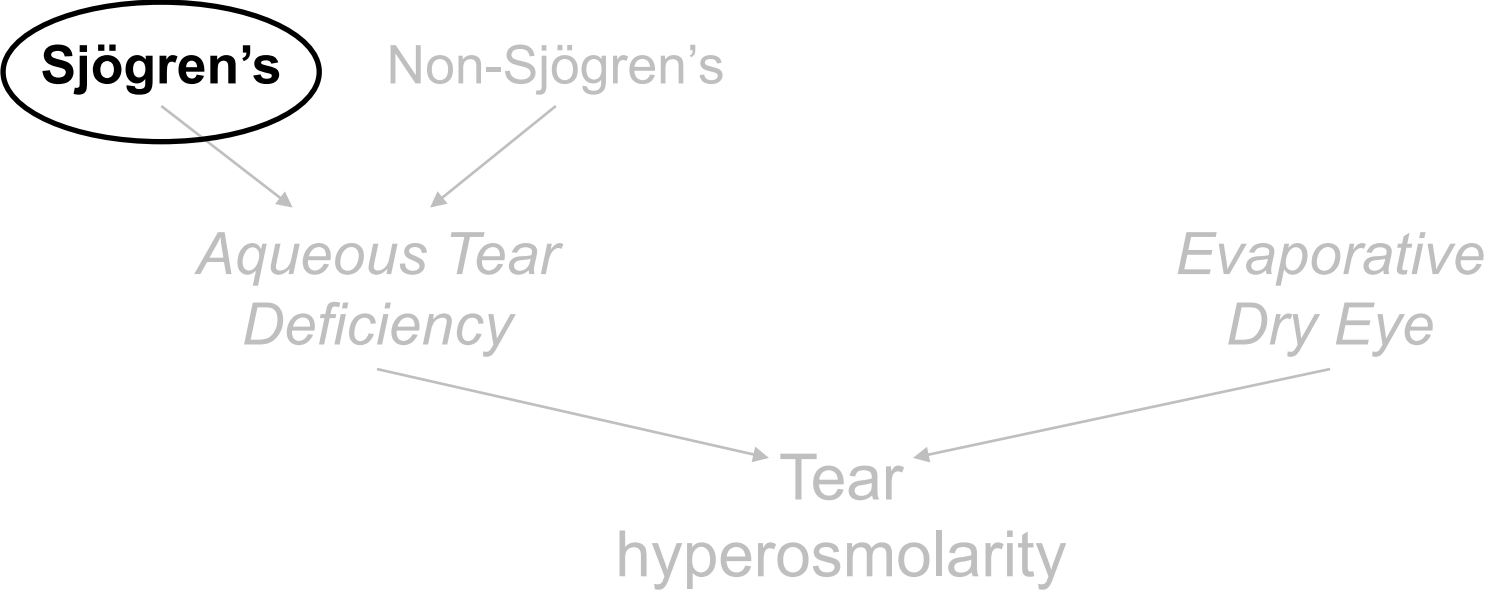
# Dry Eye Syndrome



Are the infiltrating lymphocytes T-cells, or B-cells?  
T-cells

What is Sjögren's syndrome (SS)?

A chronic autoimmune disorder characterized by **lymphocytic infiltration** of exocrine glands



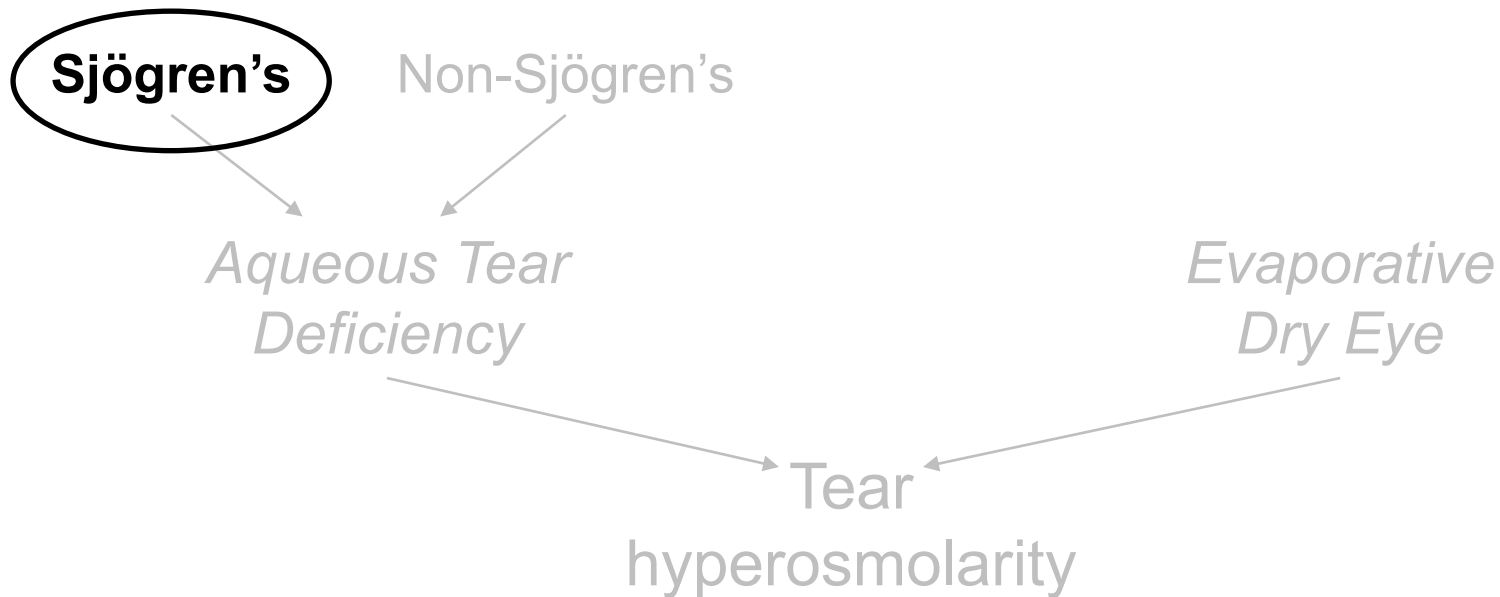
# Dry Eye Syndrome



*What is Sjögren's syndrome (SS)?*

A chronic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands

*Does SS have a gender predilection?*



# Dry Eye Syndrome

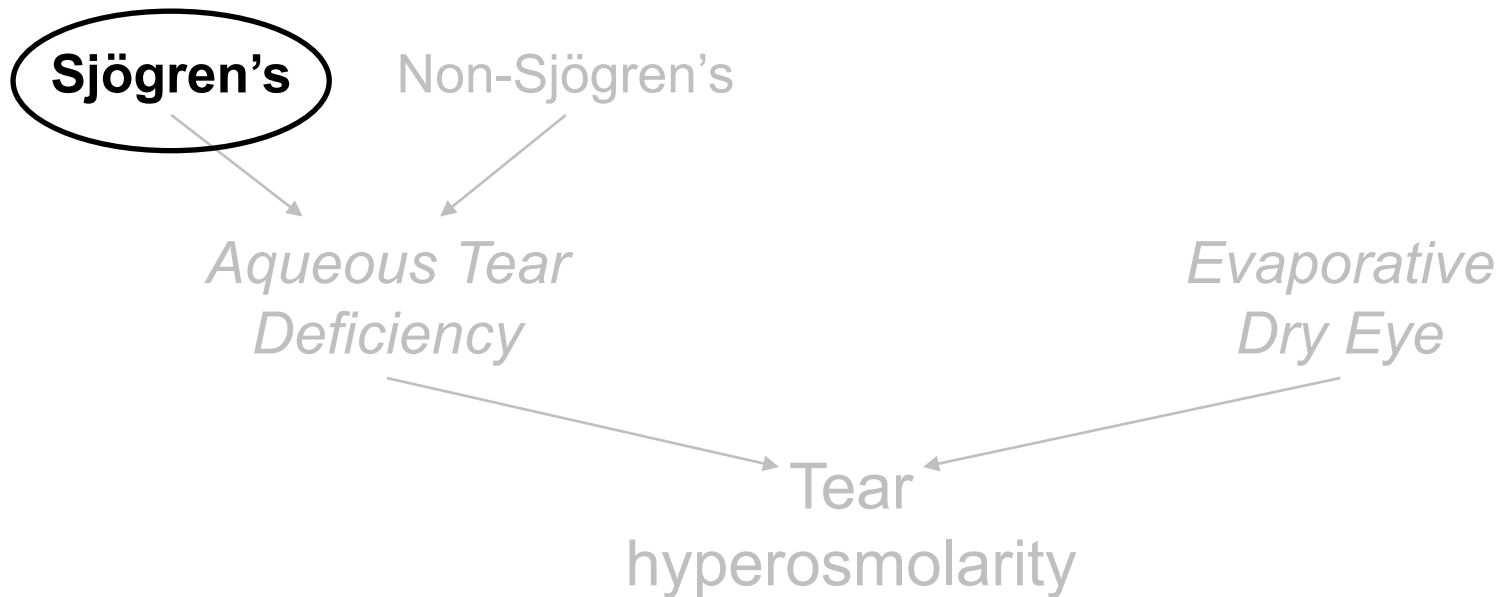


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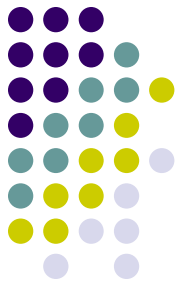
*Does SS have a gender predilection?*

Yes, the vast majority of pts are





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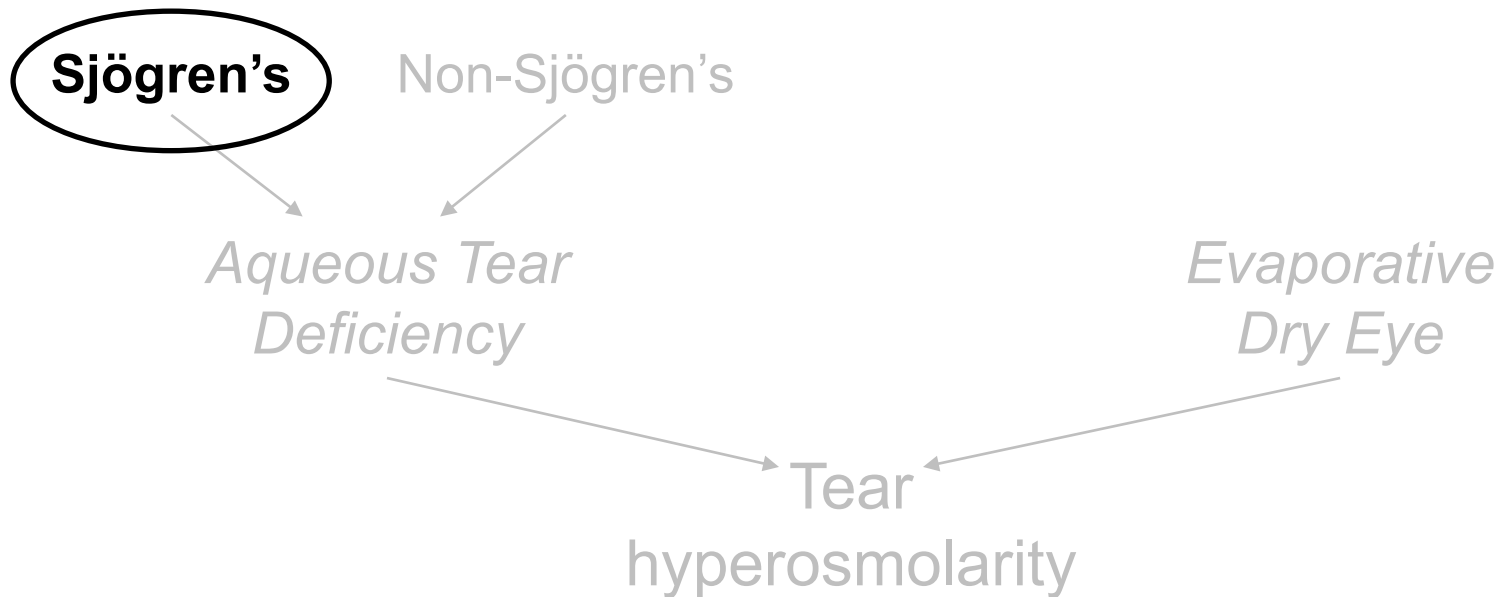


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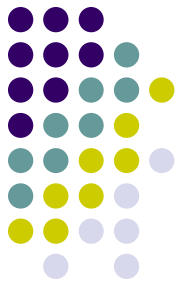
A chronic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands

*Does SS have a gender predilection?*

Yes, the vast majority of pts are female



# Dry Eye Syndrome



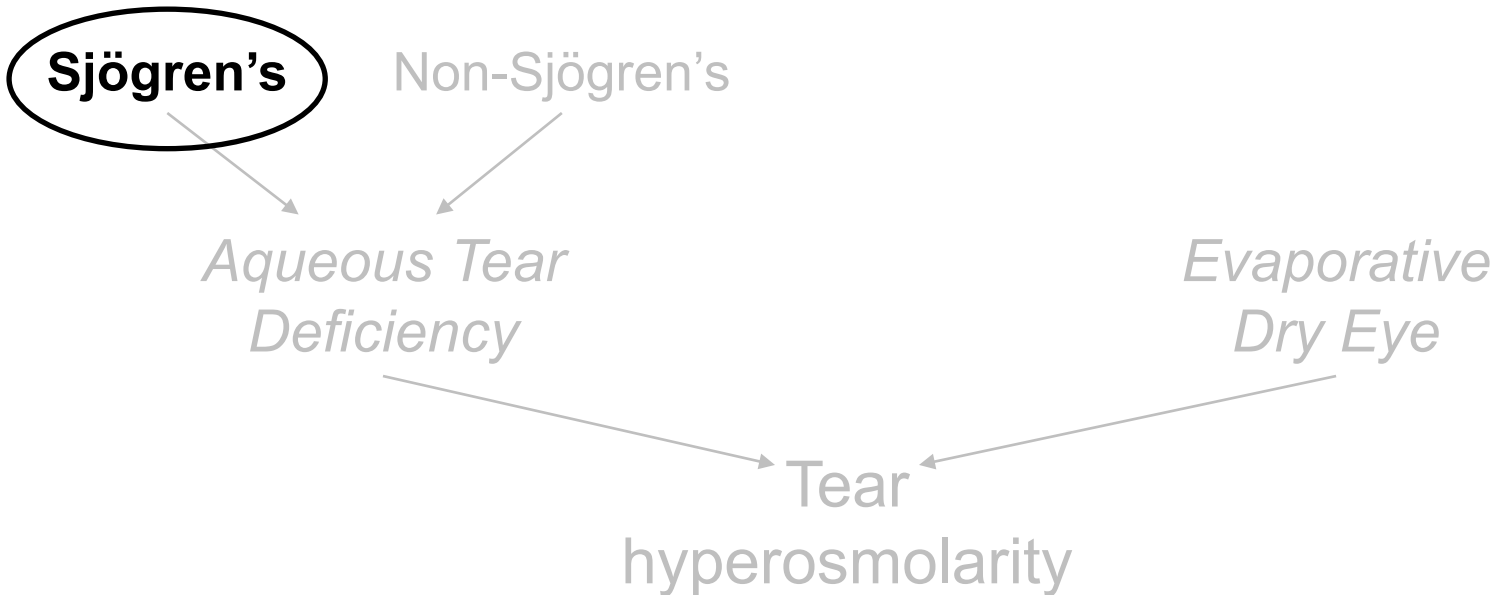
What is Sjögren's syndrome (SS)?

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SS is divided into   and   SS.



# Dry Eye Syndrome



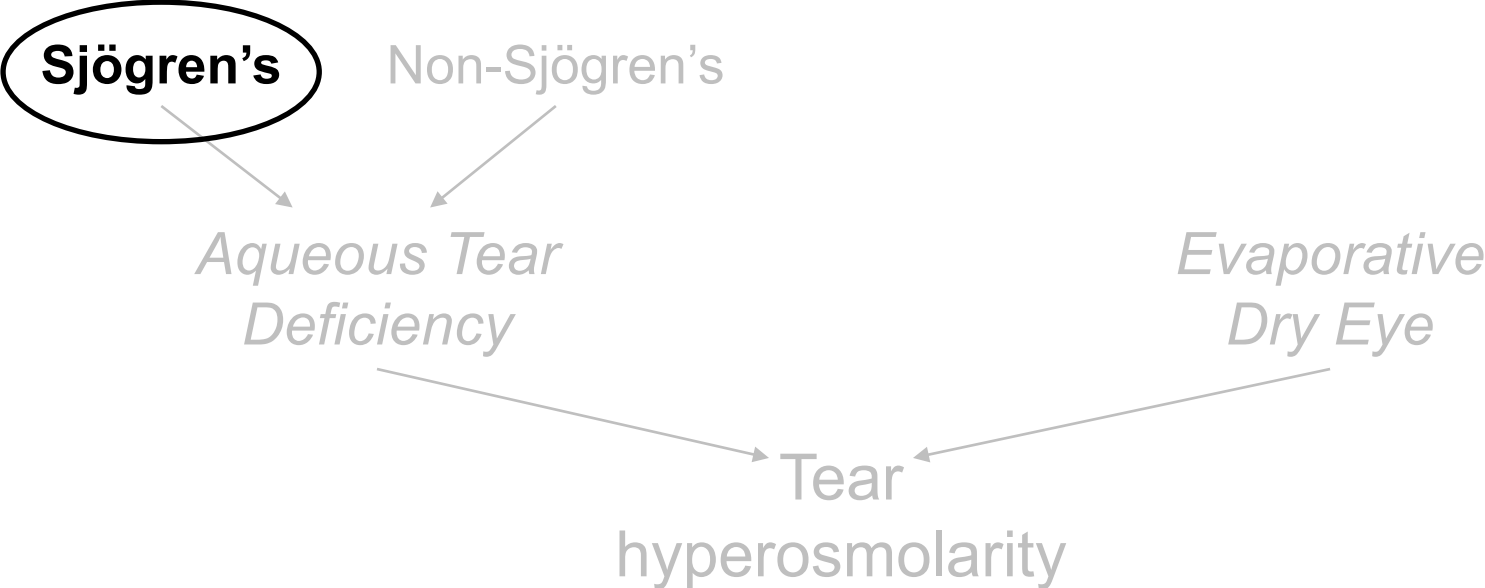
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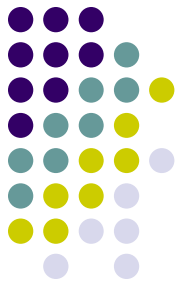
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*SS is divided into primary and secondary SS.*



# Dry Eye Syndrome



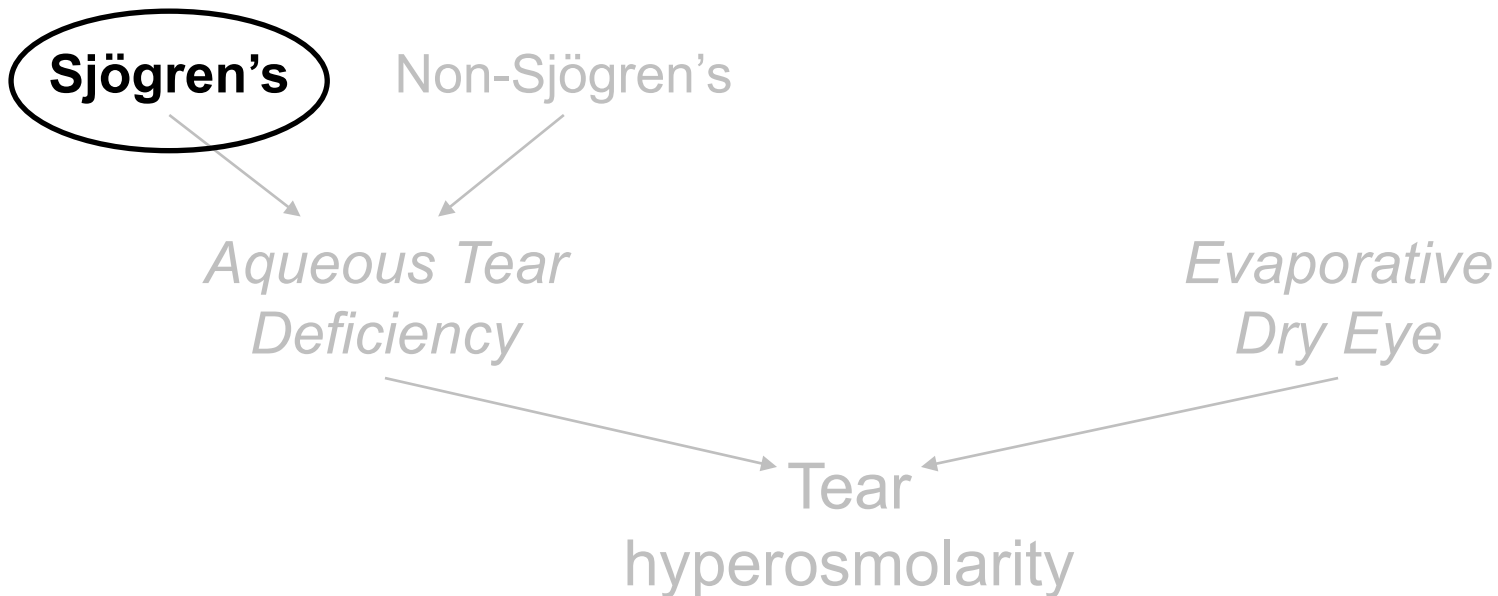
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# Dry Eye Syndrome



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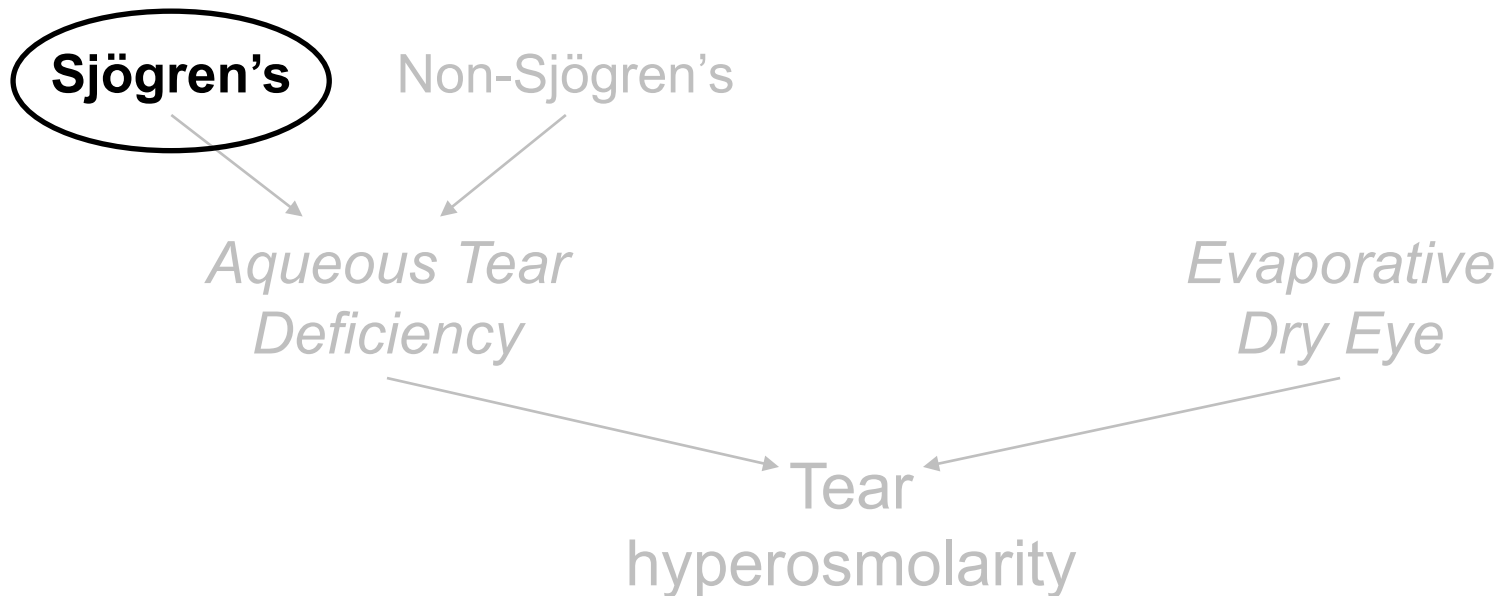
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*Does SS have a gender predilection?*

Yes, the vast majority of pts are female

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In **primary** SS, the pt does not have a systemic connective-tissue disease, whereas such a condition is present in pts with **secondary** SS



# Dry Eye Syndrome



*Speaking of lymphocytes: Pts with primary SS are at increased risk of what form of malignancy?*

*What is Sjögren's syndrome?*

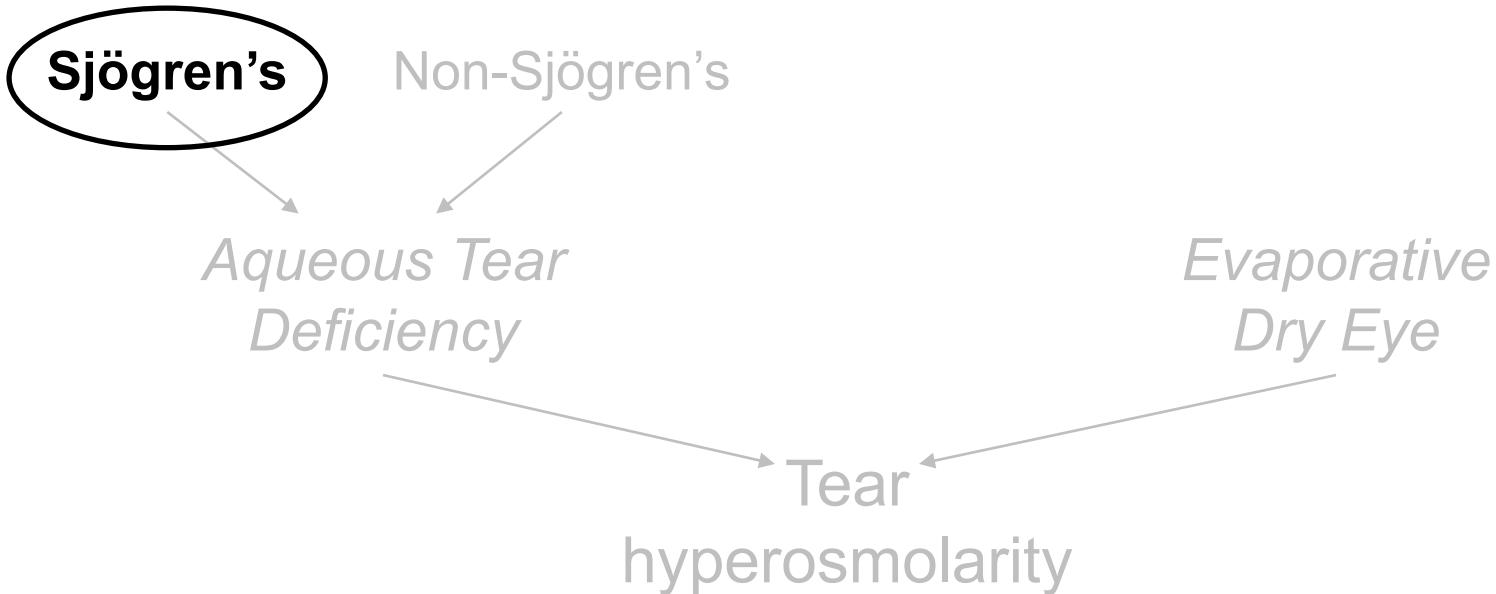
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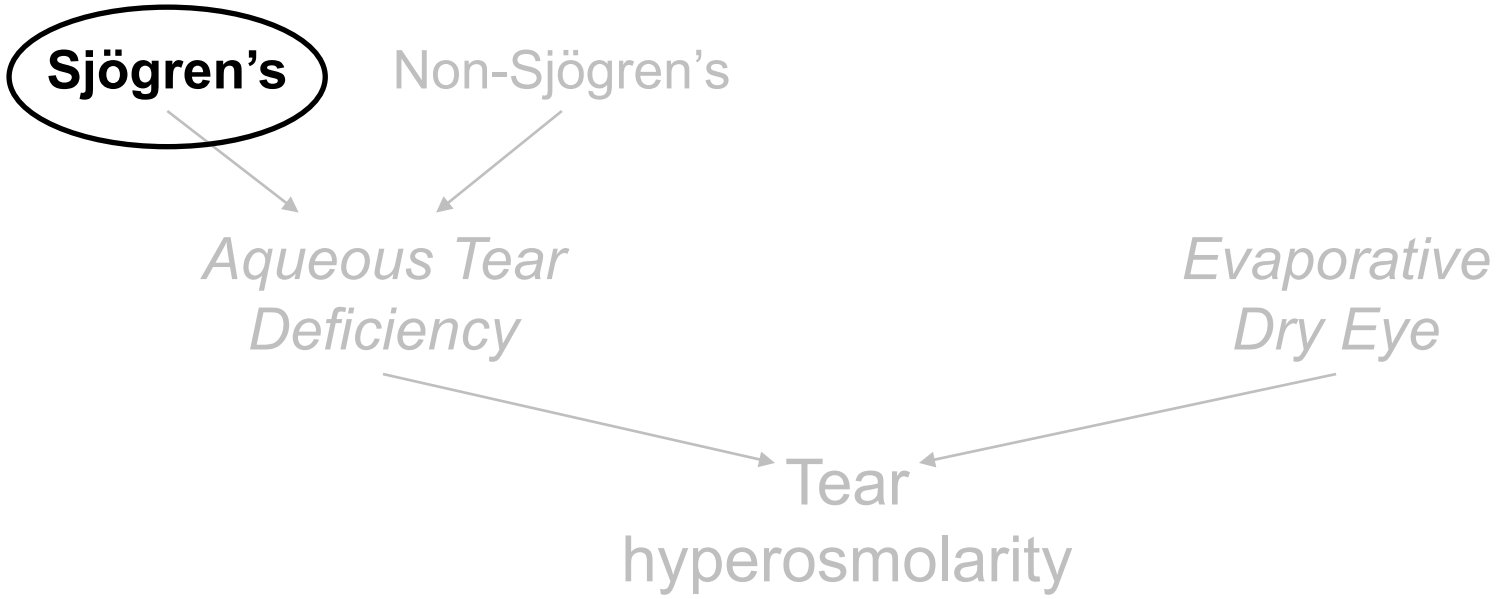
Speaking of lymphocytes: Pts with primary SS are at increased risk of what form of malignancy?  
Hodgkins vs non-lymphoma

What is Sjögren's syndrome?

A chronic autoimmune disorder characterized by **lymphocytic infiltration** of exocrine glands

Does SS have a gender predilection?  
Yes, the vast majority of pts are female

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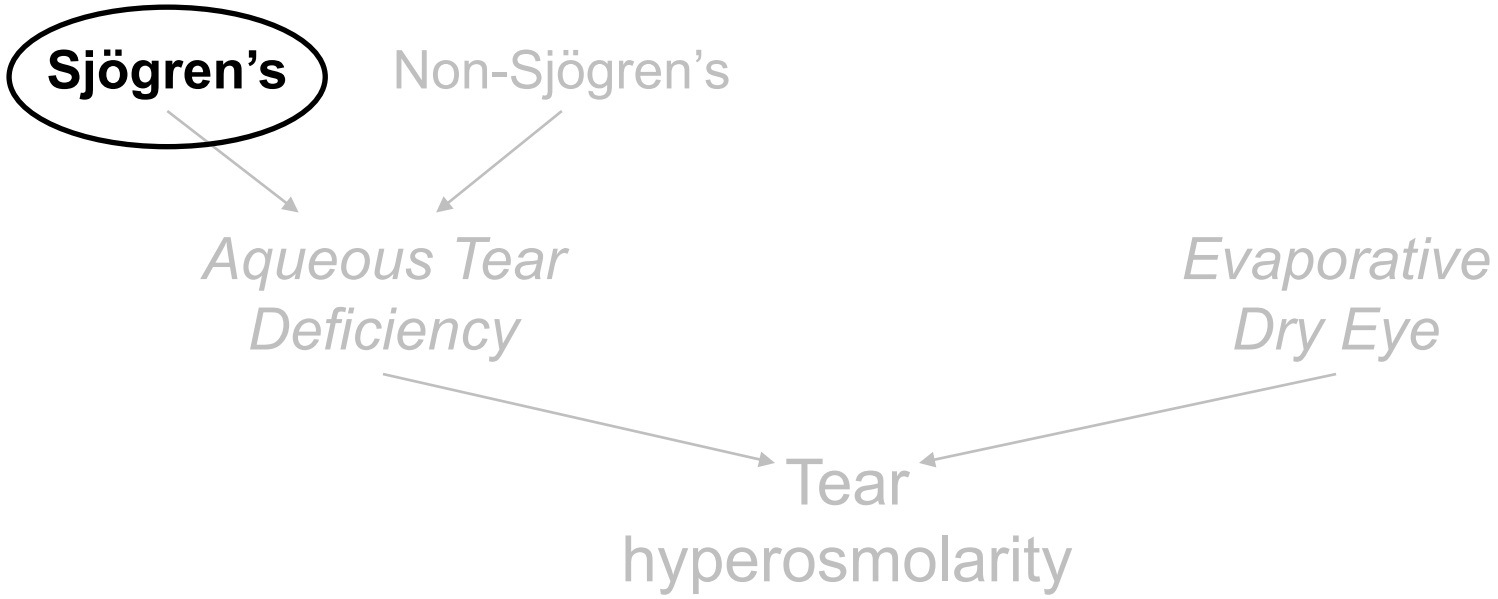
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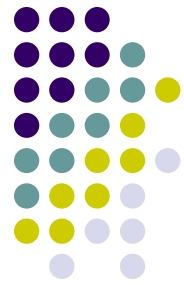
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# Dry Eye Syndrome



What is Sjögren's syndrome (SS)?

A chronic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands

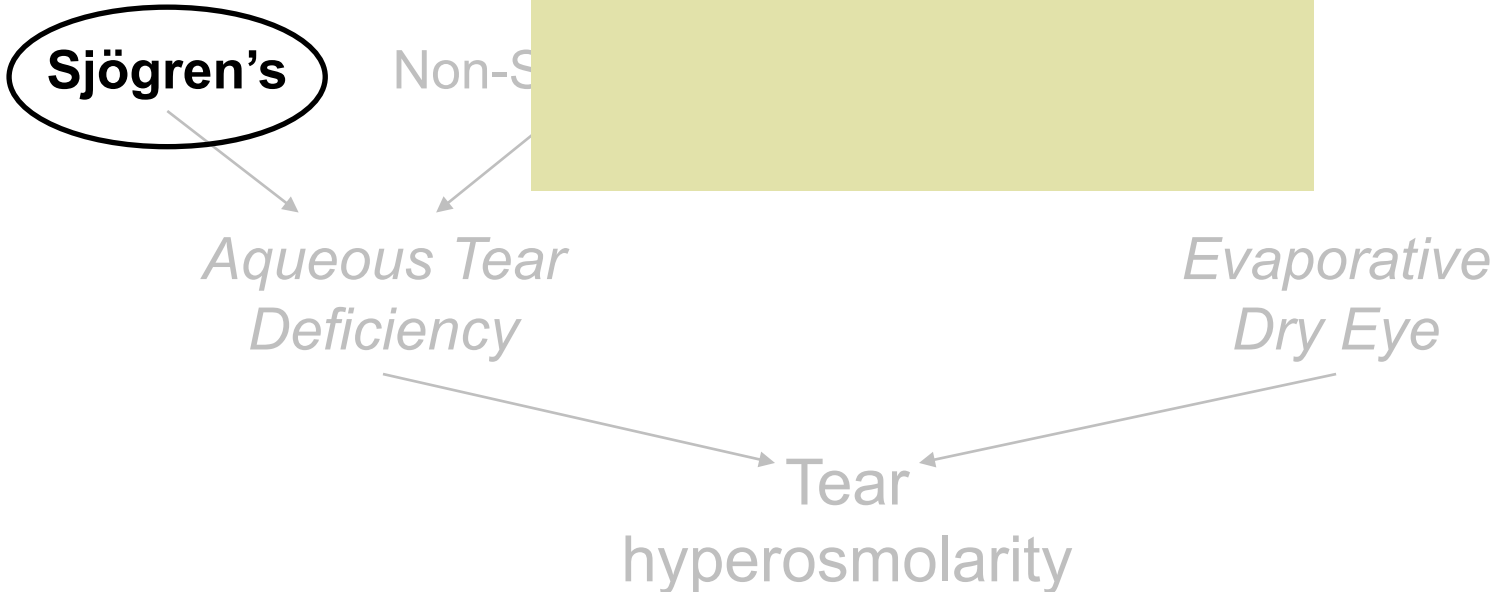
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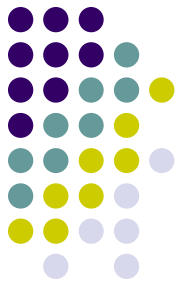
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With what CTDs is SS associated?



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With what CTDs is SS associated?  
The list is long, but the main culprits are:  
--?  
--?  
--?

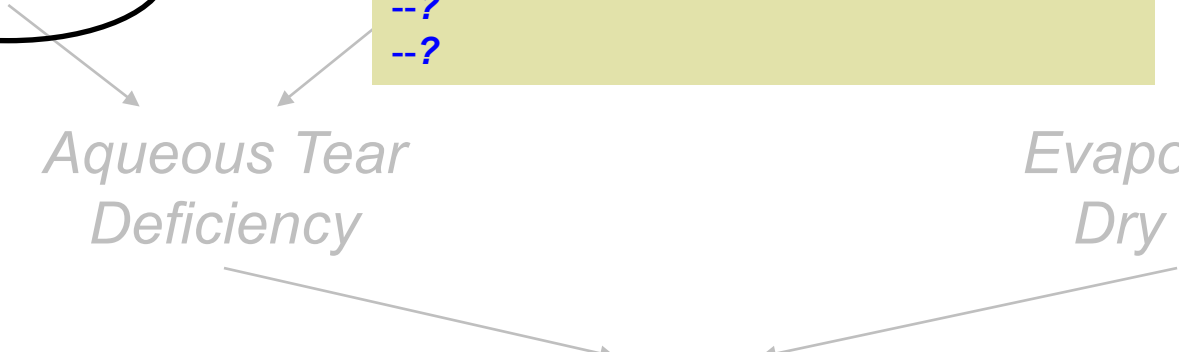
**Sjögren's**

Non-S

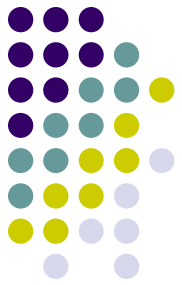
Aqueous Tear  
Deficiency

Evaporative  
Dry Eye

Tear  
hyposmolarity



# Dry Eye Syndrome



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In **primary** SS, the pt does not have **a systemic connective-tissue disease**, whereas such a condition is present in pts with **secondary** SS.

With what CTDs is SS associated?  
The list is long, but the main culprits are:  
--RA  
--SLE  
--Scleroderma (aka systemic sclerosis, SSc)

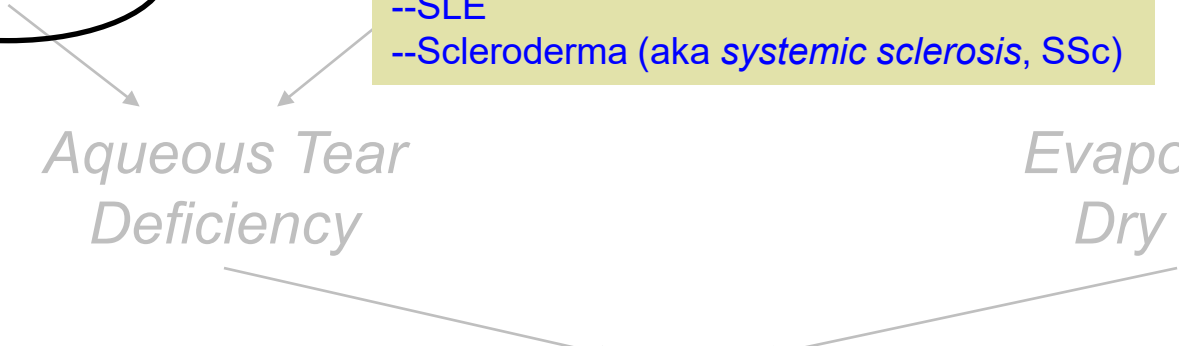
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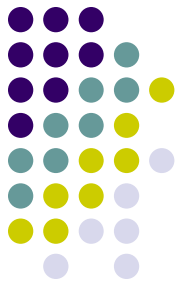
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# Dry Eye Syndrome



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In **primary** SS, the pt does not have a **systemic connective-tissue disease**, whereas such a condition is present in pts with **secondary** SS.

With what CTDs is SS associated?

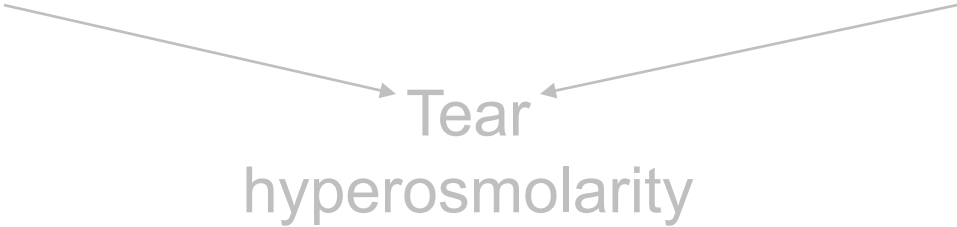
Some pts with severe Sjögren's develop enlargement of the lacrimal and parotid glands. What is the eponymous name for this condition?

--Scleroderma (aka systemic sclerosis, SSC)

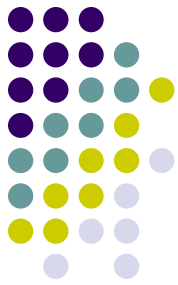
**Sjögren's**

Aqueous Tear  
Deficiency

Evaporative  
Dry Eye



# Dry Eye Syndrome



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Does SS have a gender predilection?

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SS is divided into primary and secondary SS. What's the key difference between the two?

In **primary** SS, the pt does not have a **systemic connective-tissue disease**, whereas such a condition is present in pts with **secondary** SS.

With what CTDs is SS associated?

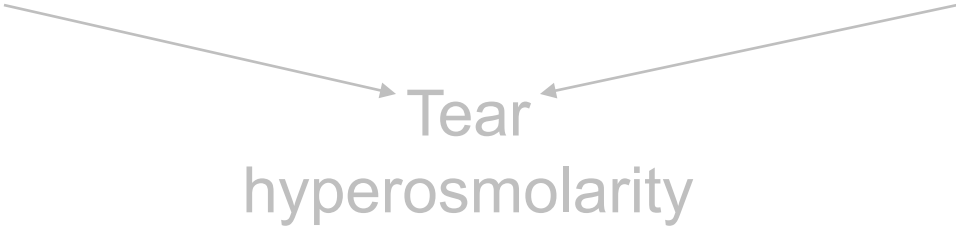
Some pts with severe Sjögren's develop enlargement of the lacrimal and parotid glands. What is the eponymous name for this condition?  
**Mikulicz syndrome**

--Scleroderma (aka systemic sclerosis, SSC)

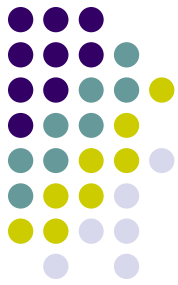
**Sjögren's**

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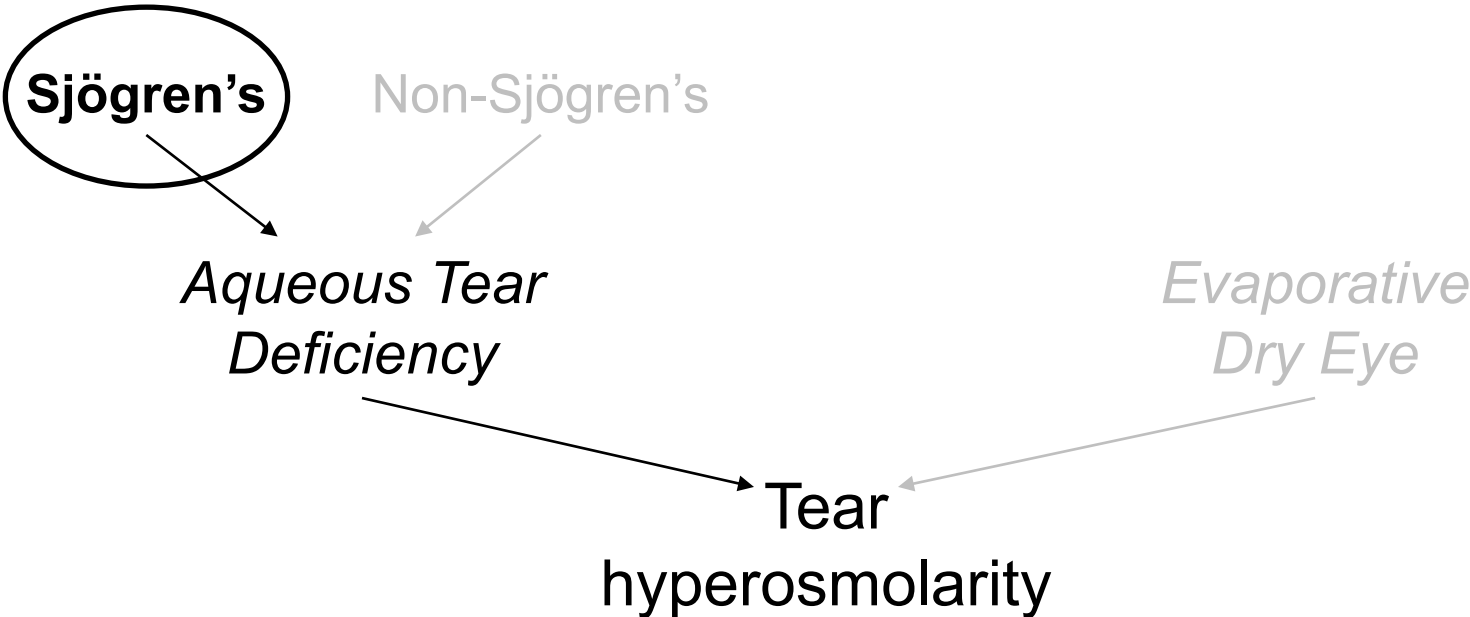


# Dry Eye Syndrome



*In SS, aqueous hyposecretion (and therefore ATD) results from autoimmune-mediated lymphocytic infiltration of the lacrimal glands.*

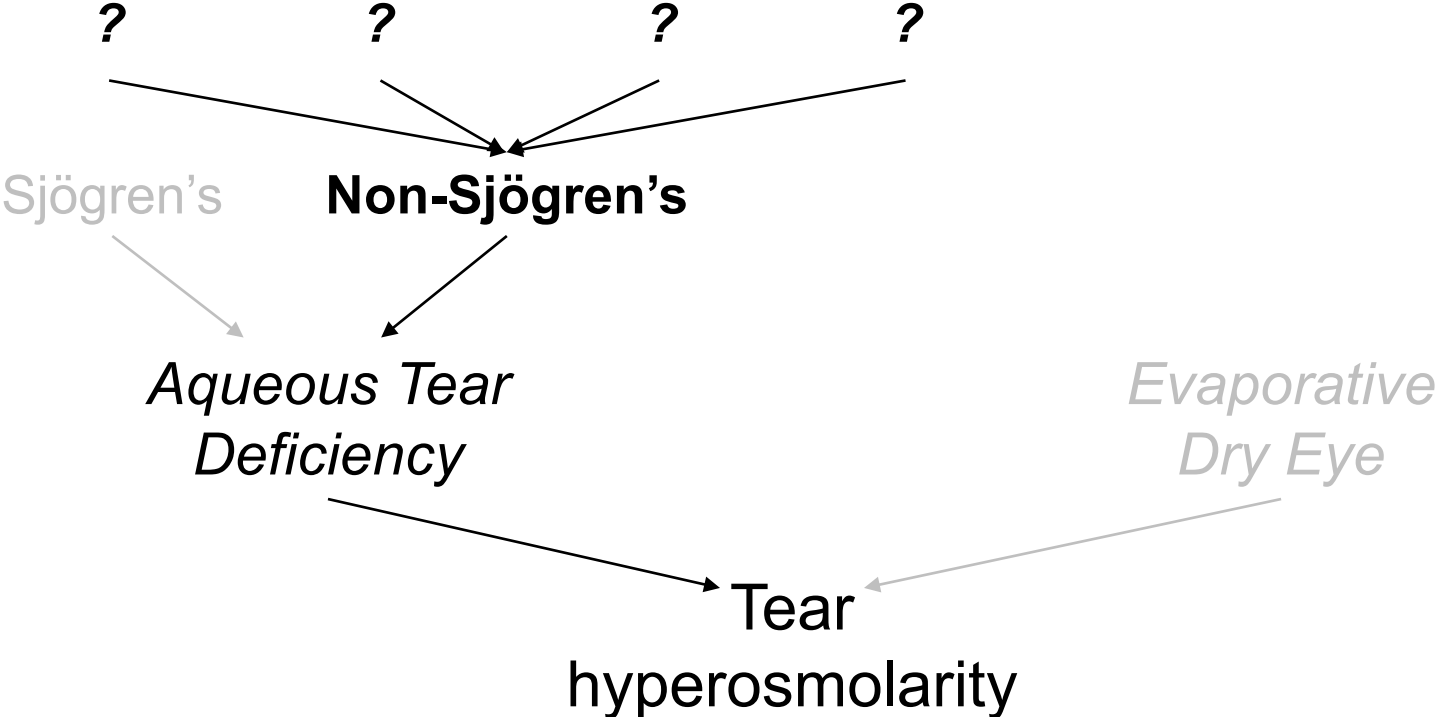
*(Statement of fact—not a question. Keep going.)*



# Dry Eye Syndrome



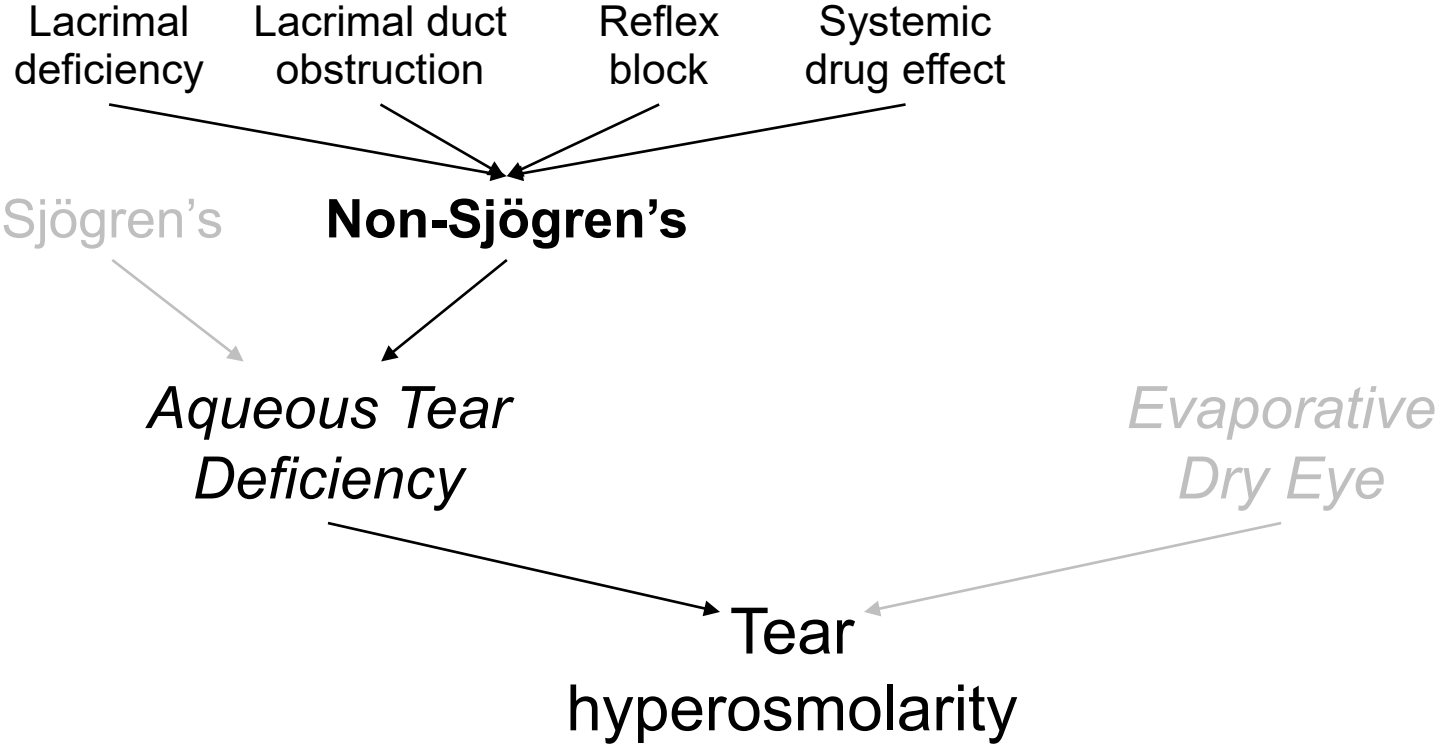
*In SS, aqueous hyposecretion (and therefore ATD) results from autoimmune-mediated lymphocytic infiltration of the lacrimal glands. In **non-Sjögren's** ATD, four broad categories of conditions leading to lacrimal gland hyposecretion have been identified. What are they?*



# Dry Eye Syndrome



*In SS, aqueous hyposecretion (and therefore ATD) results from autoimmune-mediated lymphocytic infiltration of the lacrimal glands. In **non-Sjögren's** ATD, four broad categories of conditions leading to lacrimal gland hyposecretion have been identified. What are they?*

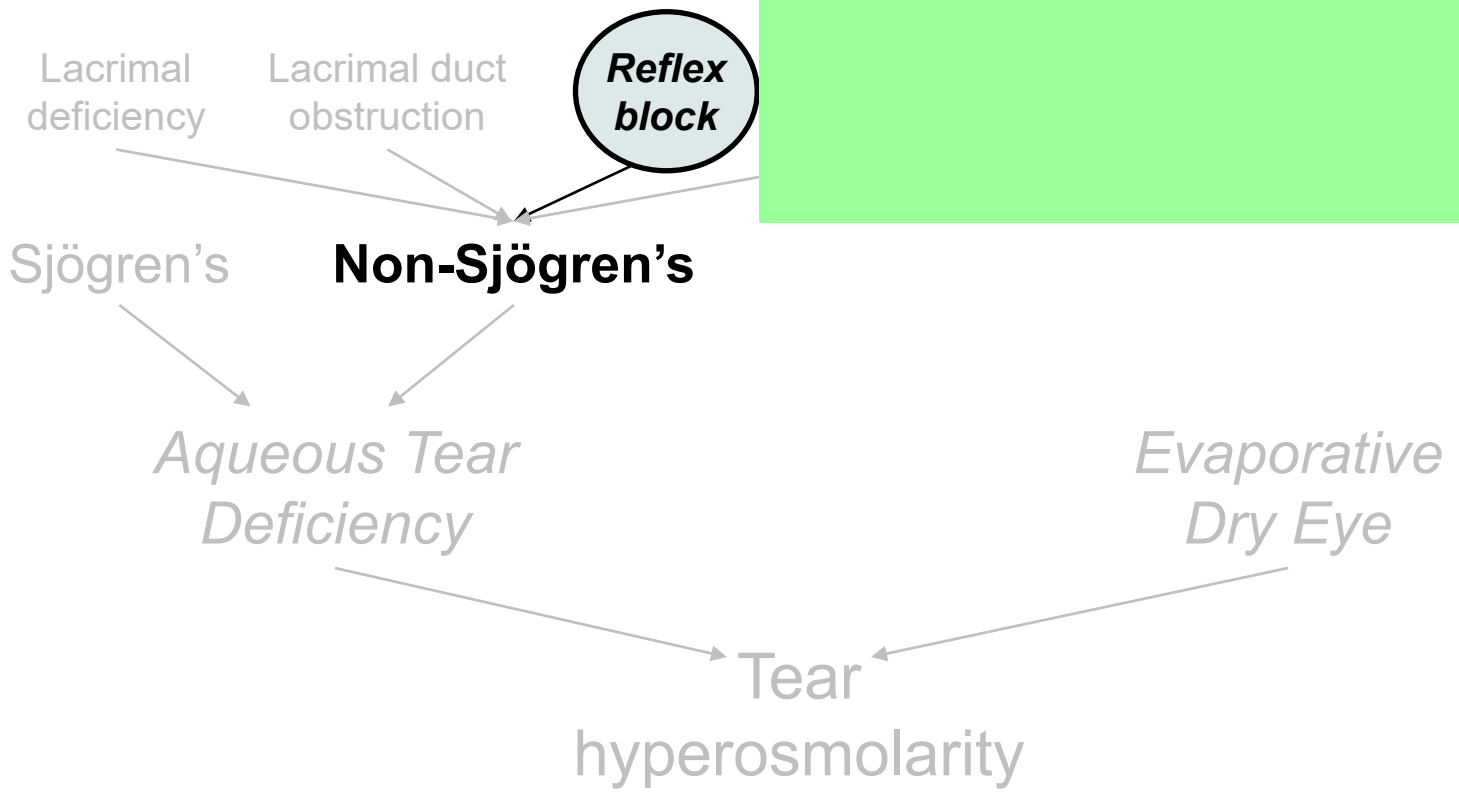




# Dry Eye

What does reflex block mean?

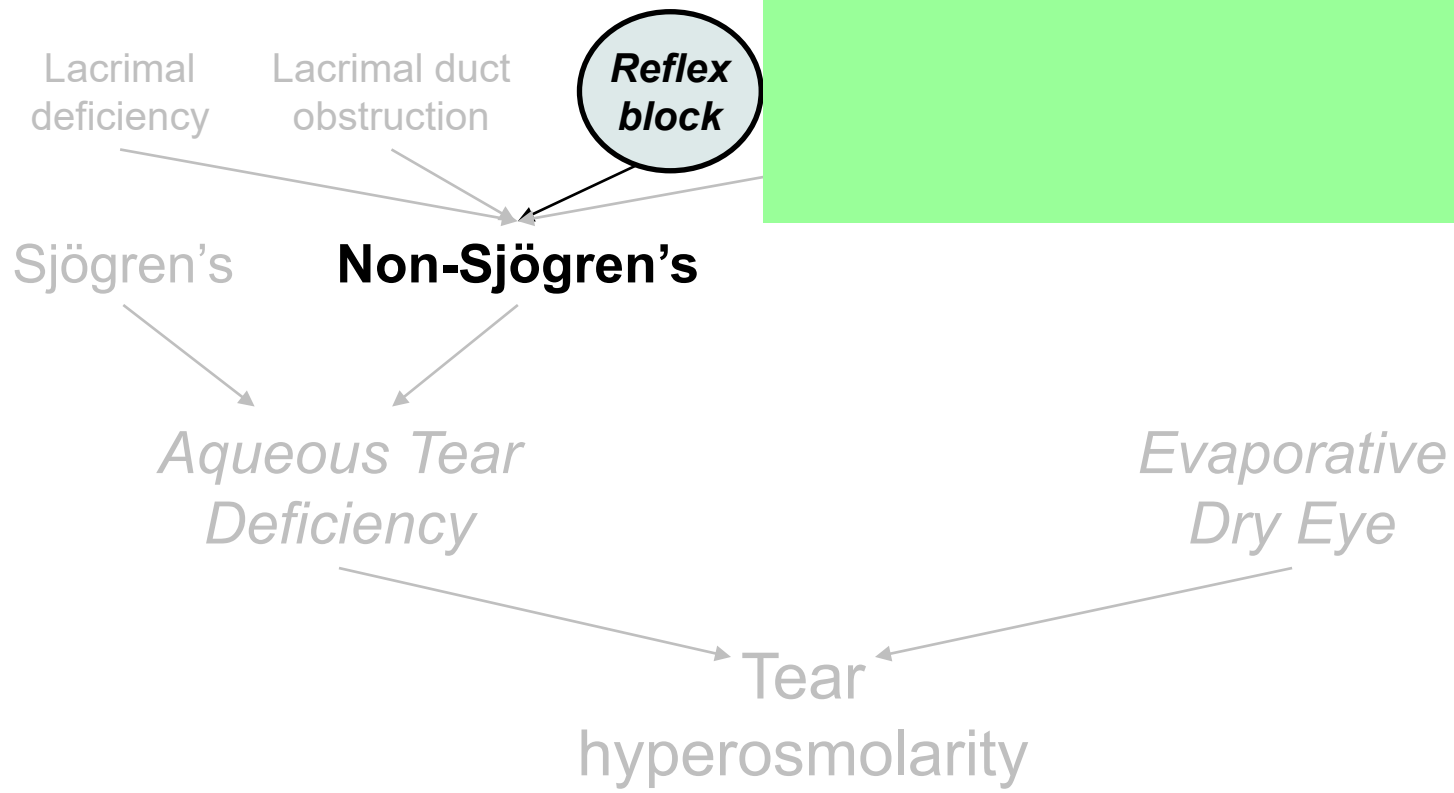
In SS, aqueous hyposecretion (and therefore mediated lymphocytic infiltration of the lacrimal gland) has led to four broad categories of conditions leading to dry eye. What are they?



# Dry Eye

What does reflex block mean?  
Recall that tear production is considered largely reflexive. Thus, any break in the LFU reflex circuit will lead to ATD.

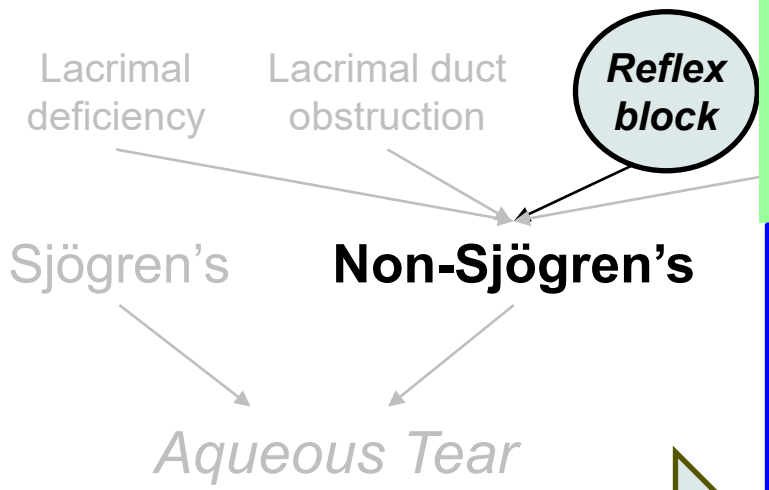
*In SS, aqueous hyposecretion (and therefore mediated lymphocytic infiltration of the lacrimal gland) has been identified. What are the four broad categories of conditions leading to ATD that have been identified. What are they?*



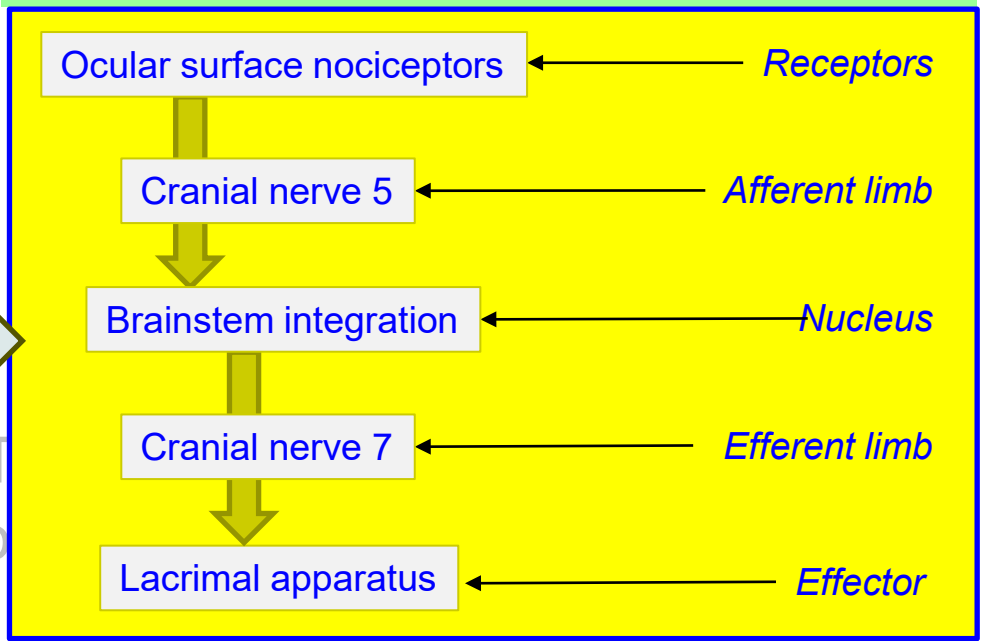
# Dry Eye

What does reflex block mean?  
Recall that tear production is considered largely reflexive. Thus, any break in the LFU reflex circuit will lead to ATD.

In SS, aqueous hyposecretion (and therefore mediated lymphocytic infiltration of the lacrimal gland) has been identified. What are they?



Here's a no-frills version of the LFU for reference



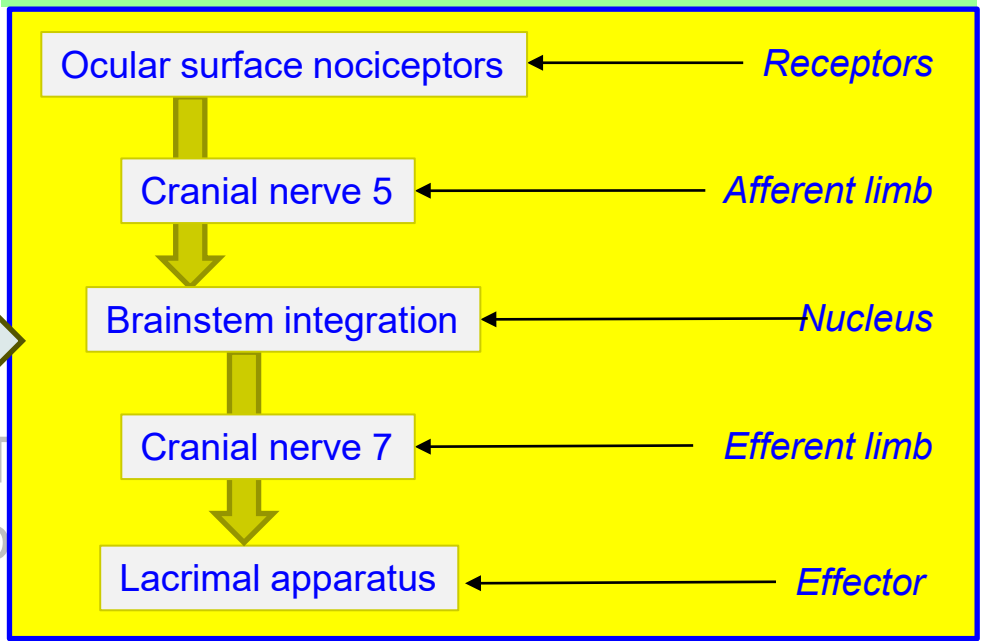
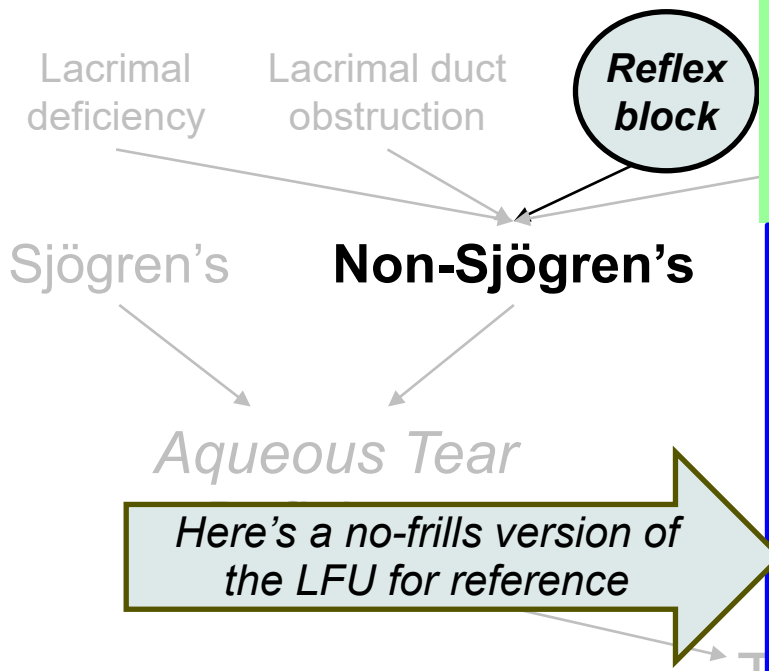
hypero

# Dry Eye

What does reflex block mean?  
Recall that tear production is considered largely reflexive. Thus, any break in the LFU reflex circuit will lead to ATD.

What are some of the common mechanisms producing afferent limb block?

In SS, aqueous hyposecretion (and therefore mediated lymphocytic infiltration of the lacrimal gland) has been identified. What are they?



hypero

# Dry Eye

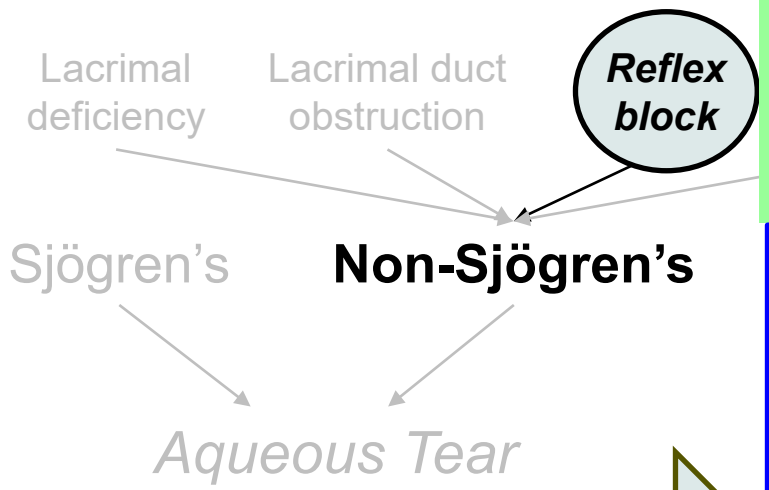
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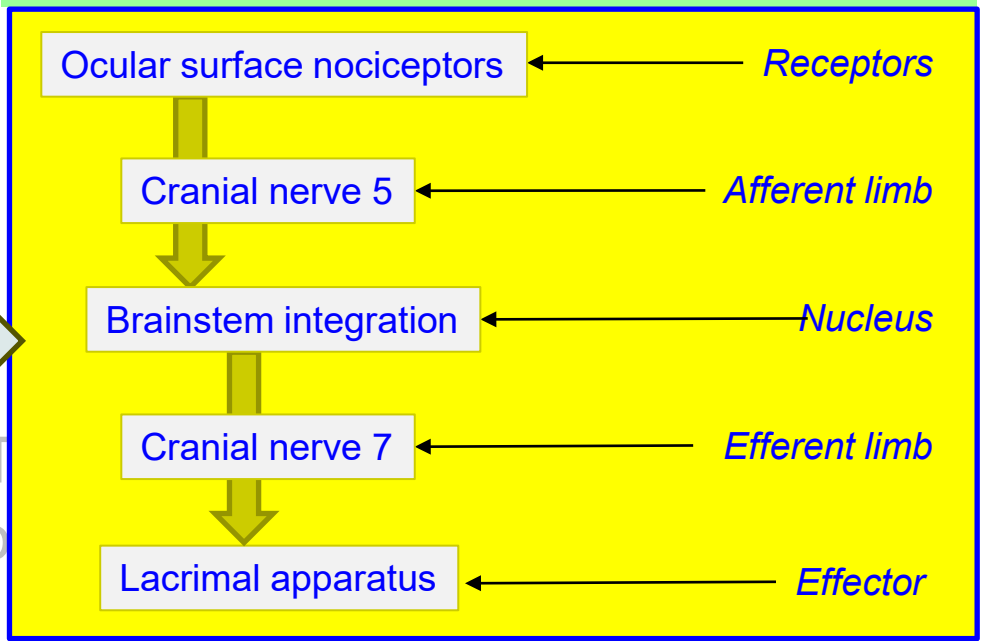
--The most common culprits are conditions leading to

two words

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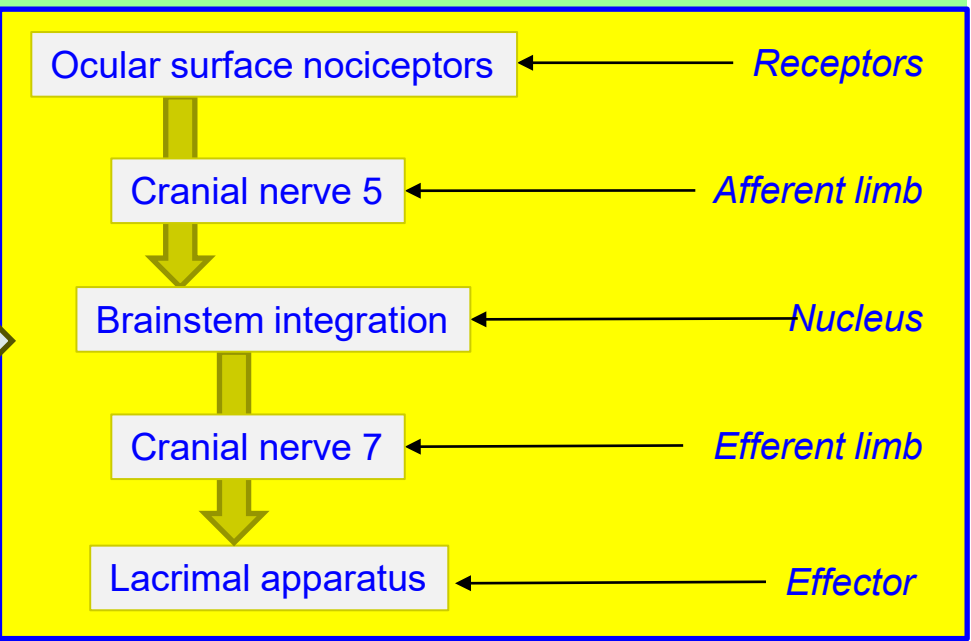
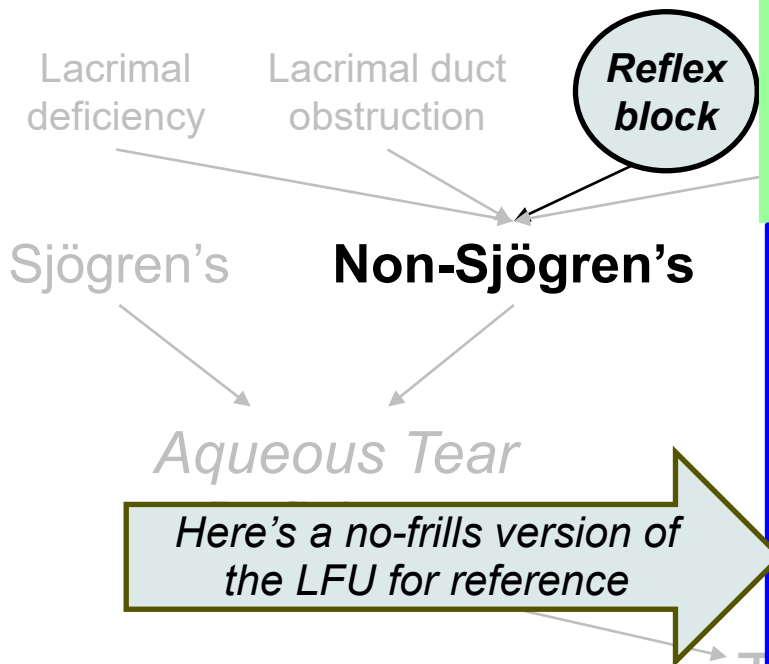
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hypero

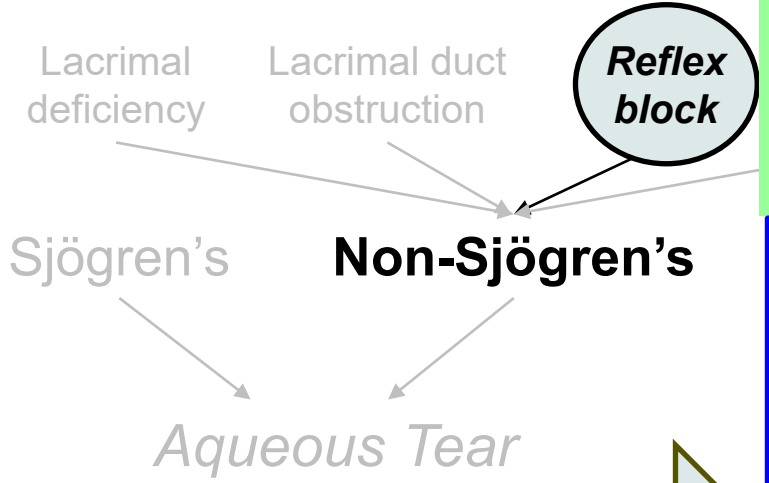
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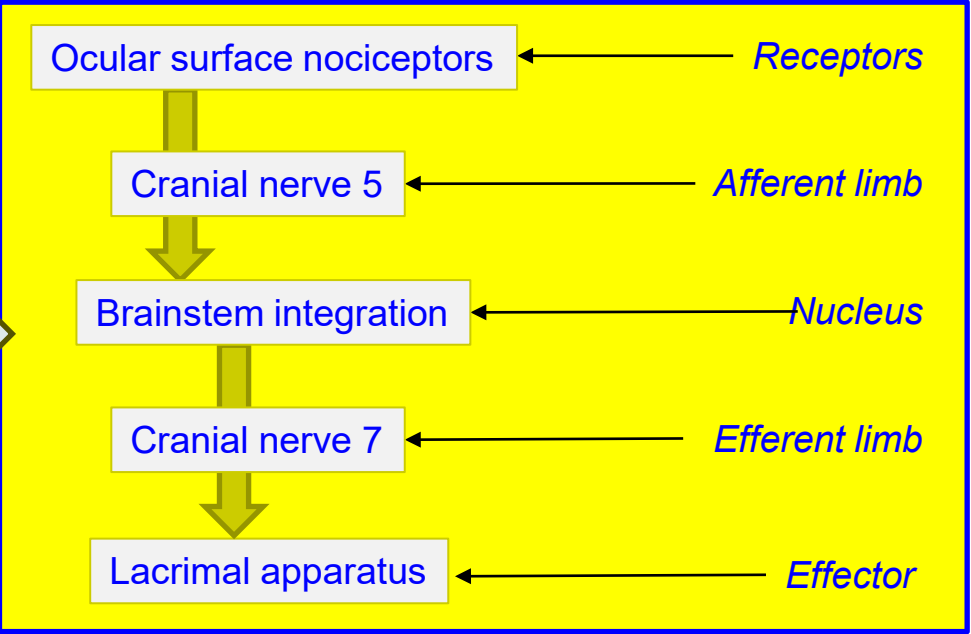
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  - ?
  - ?
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hypero

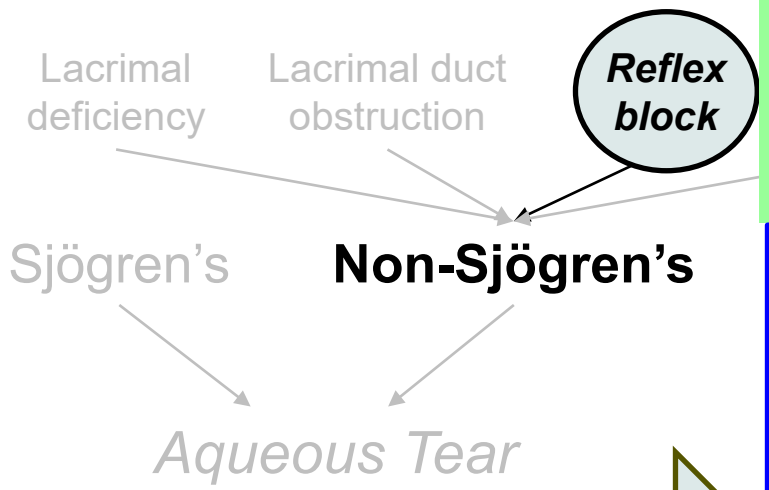
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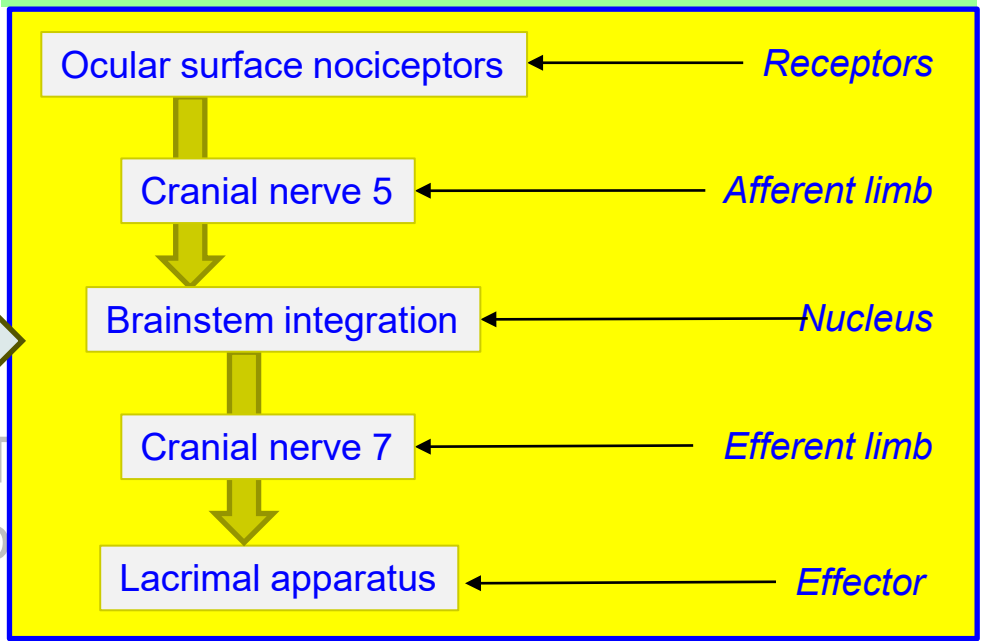
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- [Yellow box] cornea
- ?
- ?
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hypero



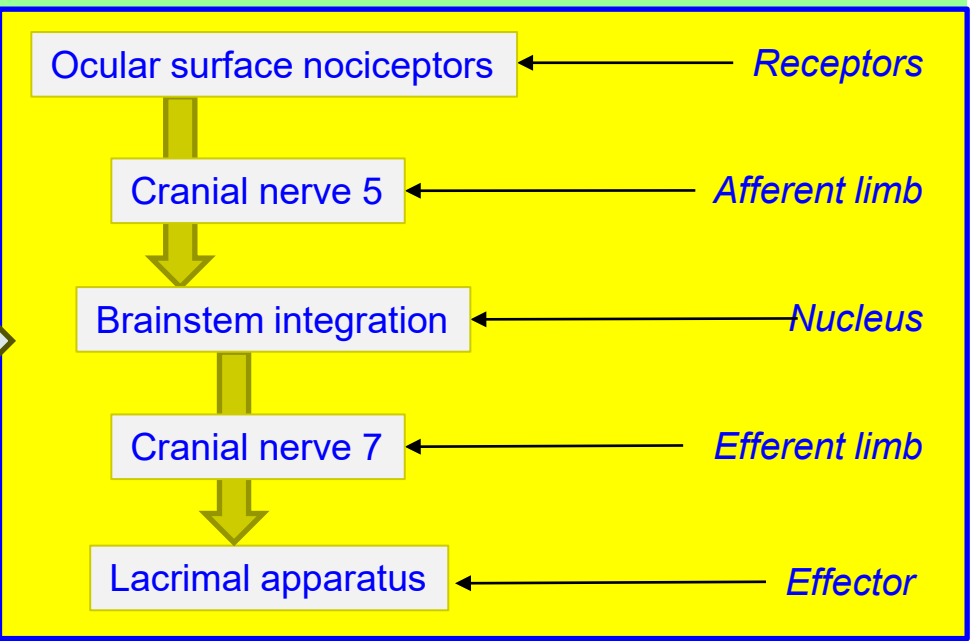
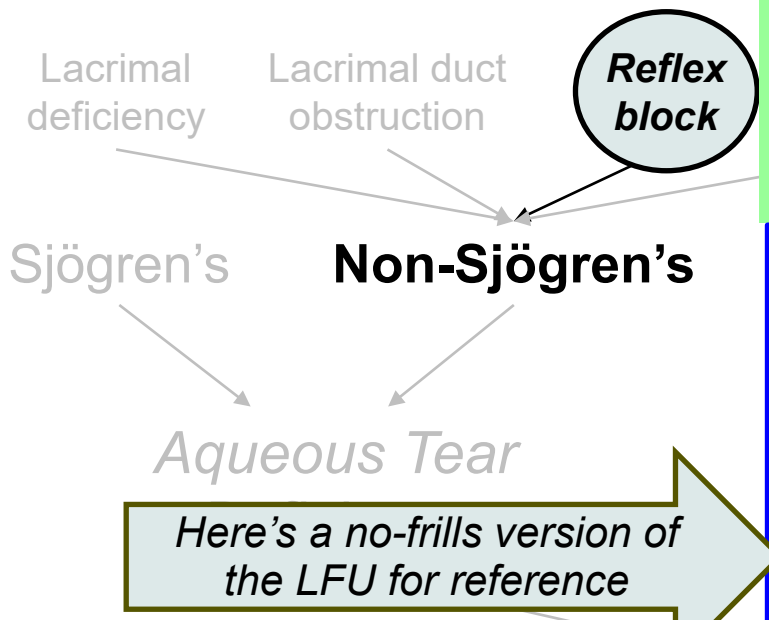
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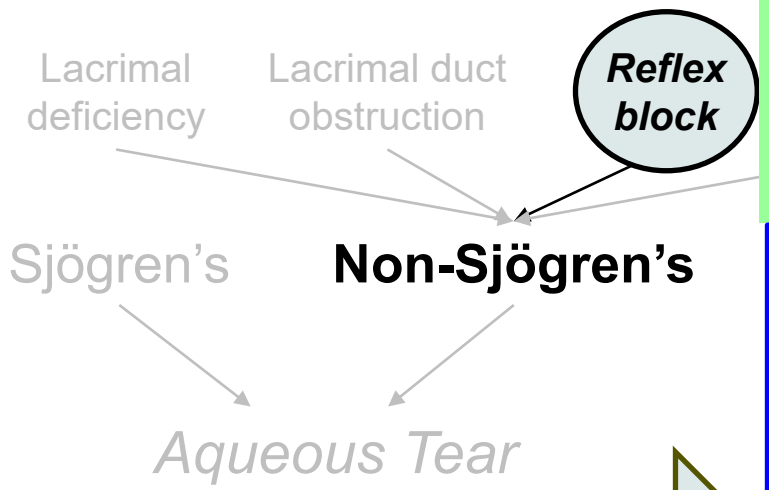
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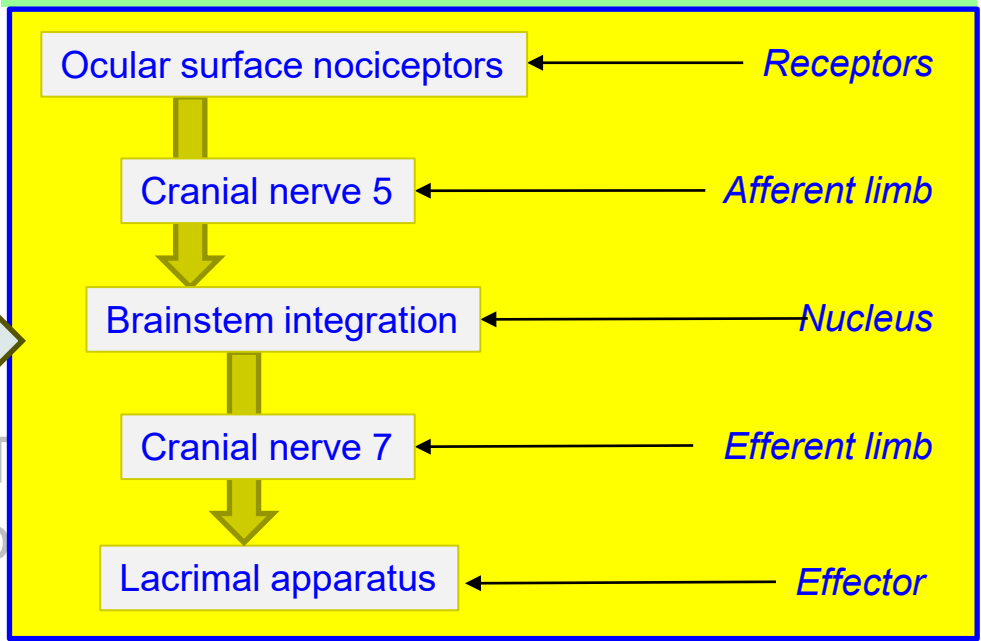
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 ----Corneal [redacted]  
 ----?  
 ----?

*In SS, aqueous hyposecretion (and therefore mediated lymphocytic infiltration of the lacrimal gland) has been identified. What are the four broad categories of conditions leading to this? What have been identified. What are they?*



*Here's a no-frills version of the LFU for reference*



hypero

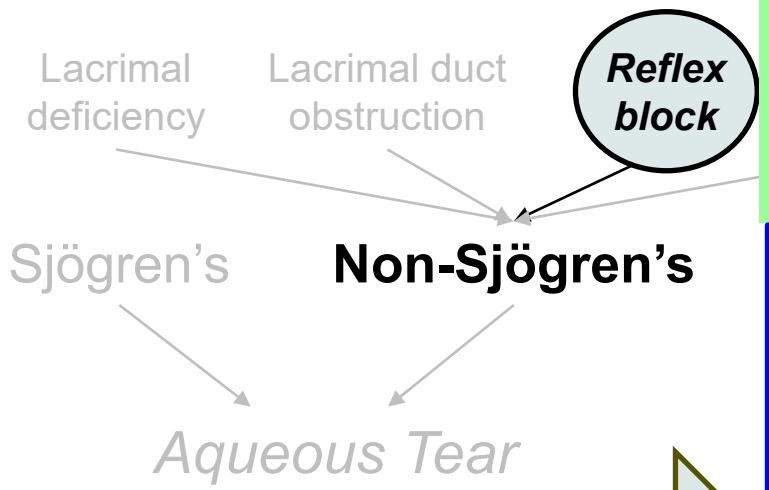
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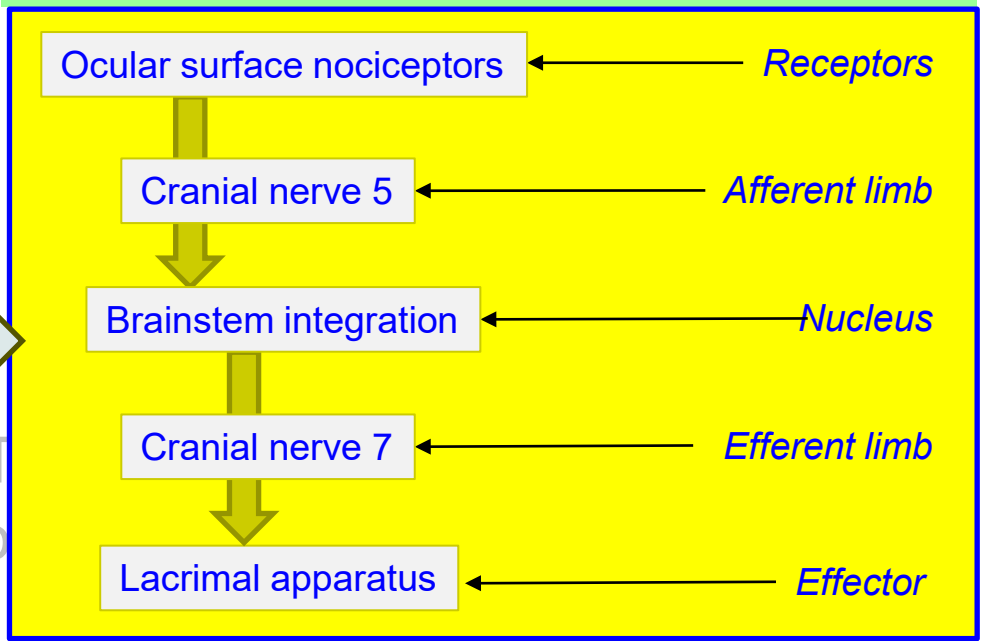
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hypero

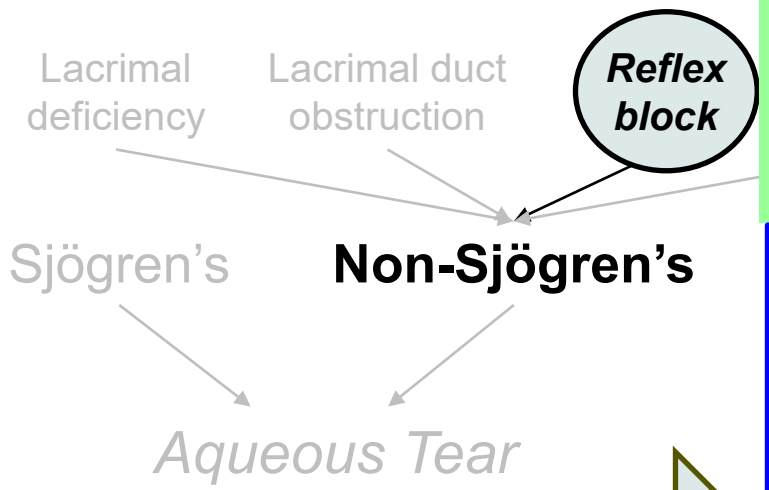
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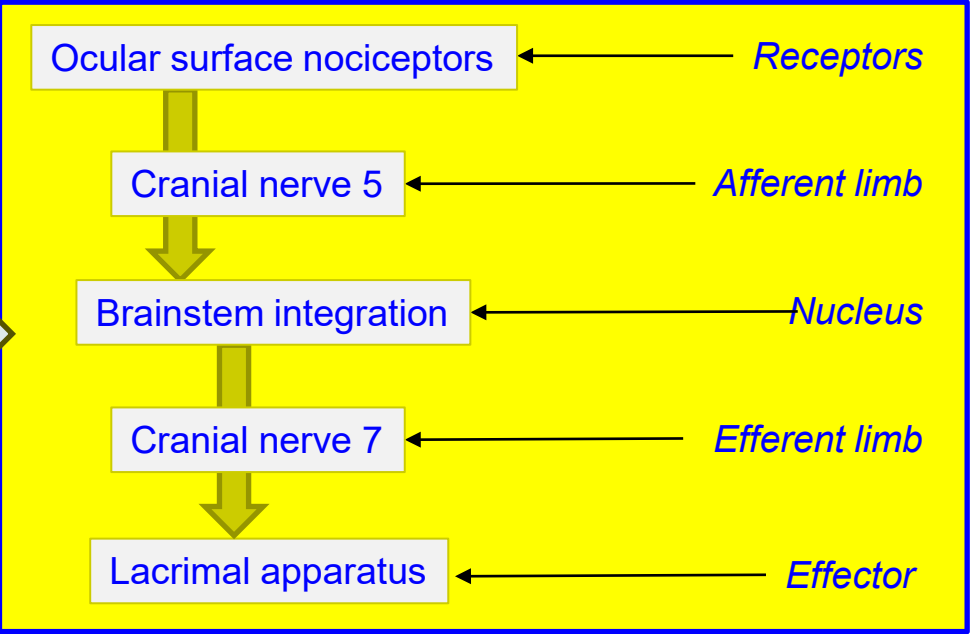
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hypero

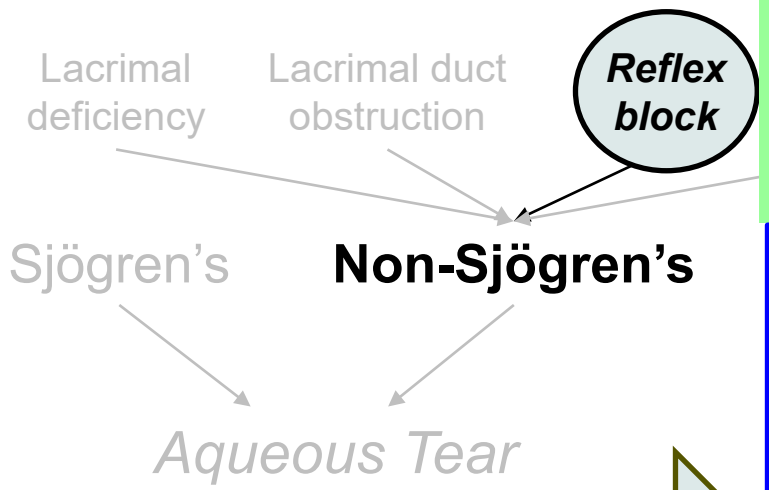
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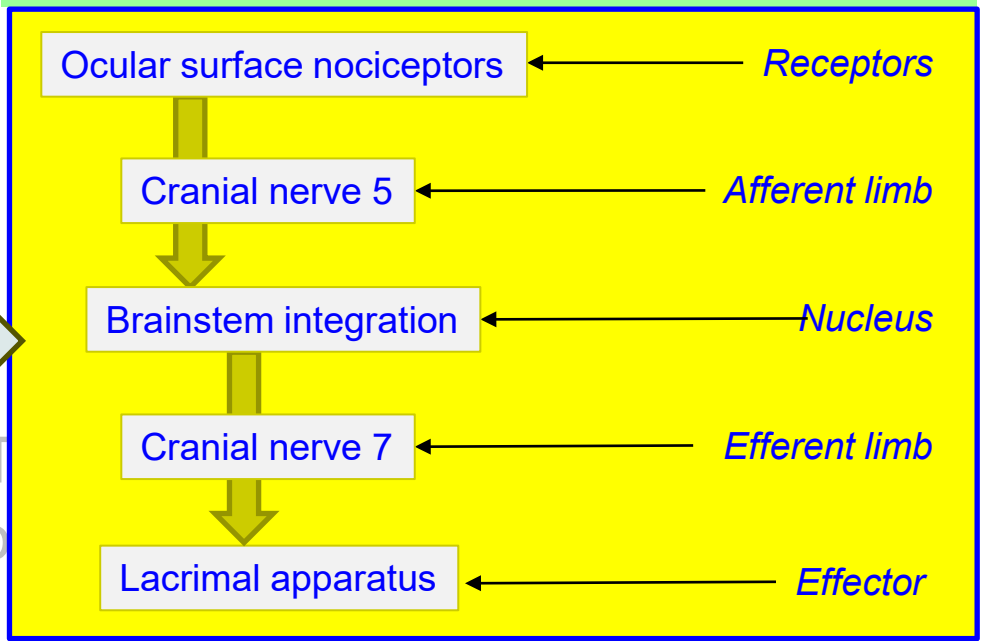
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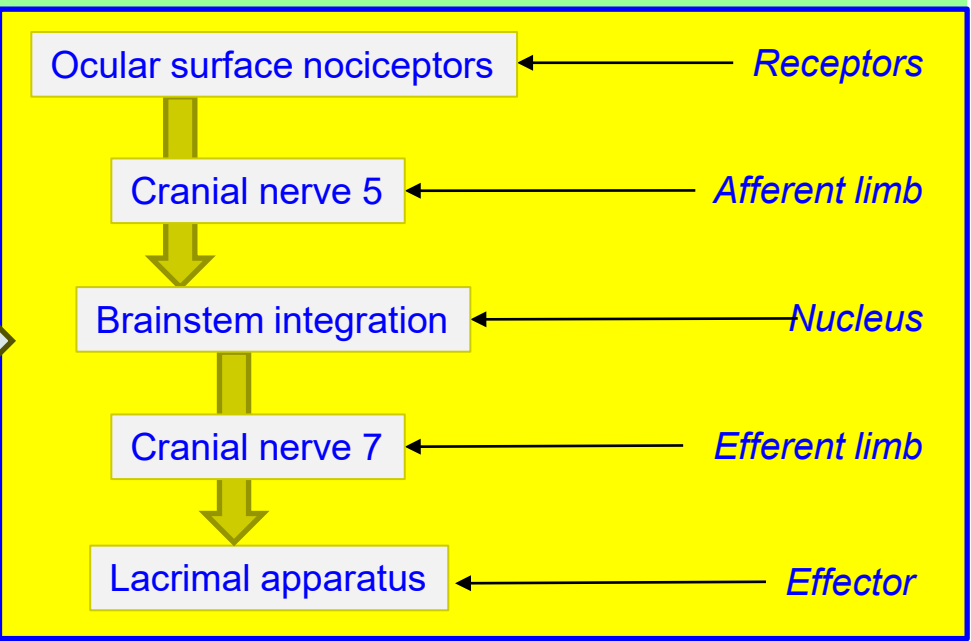
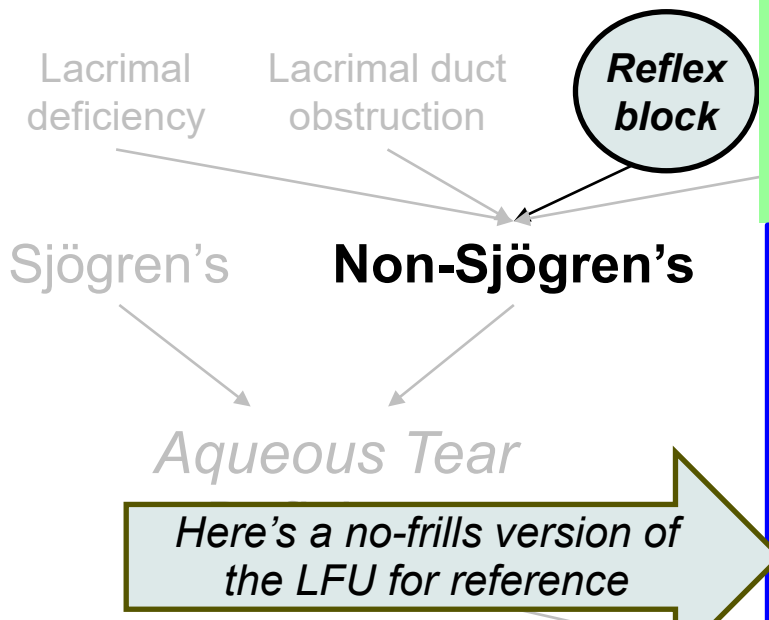
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hypero

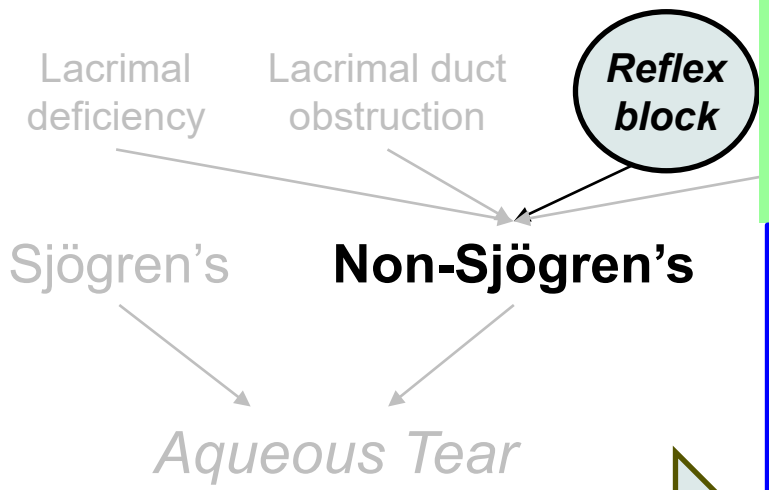
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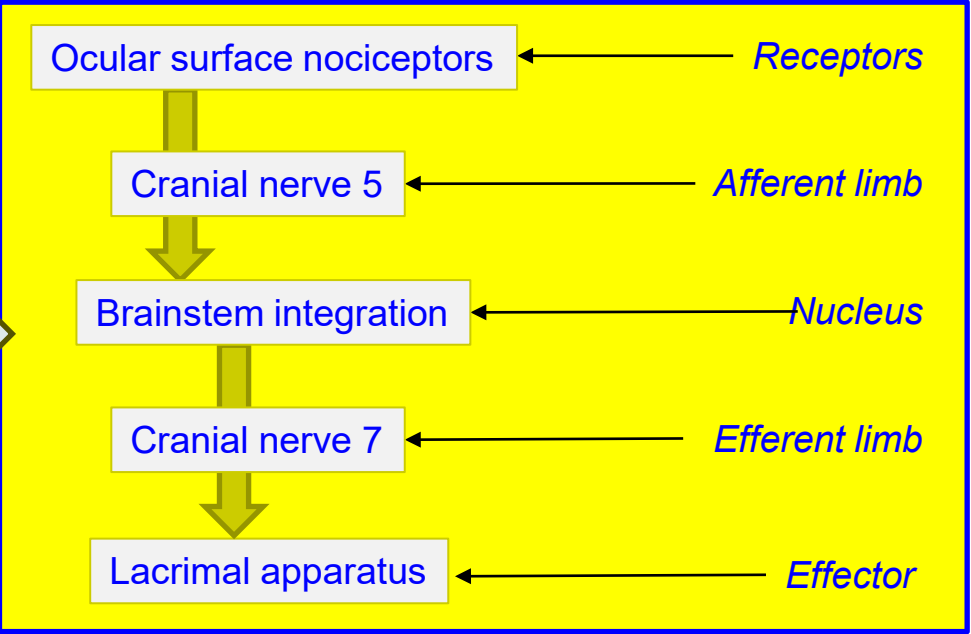
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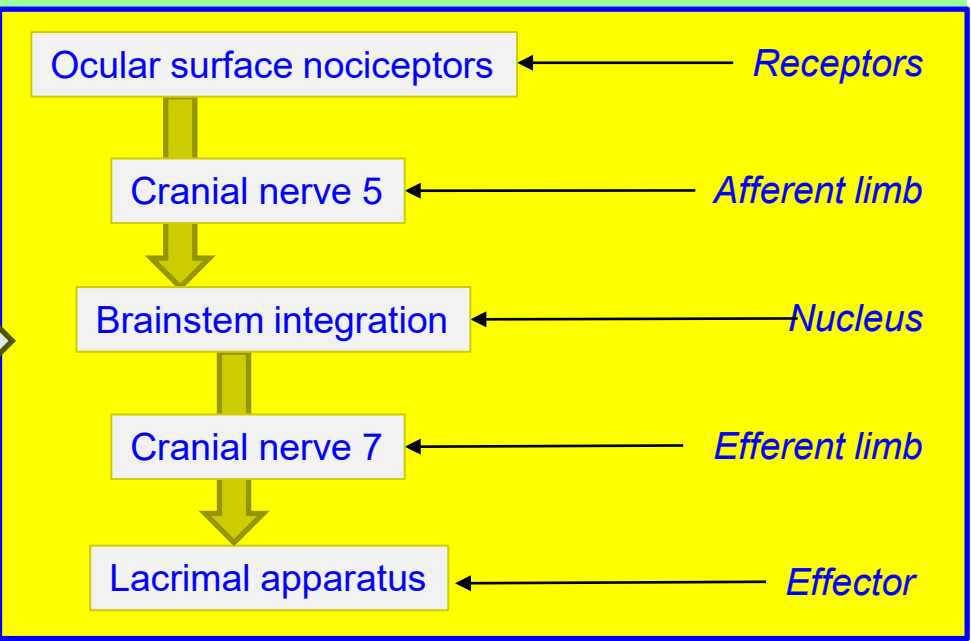
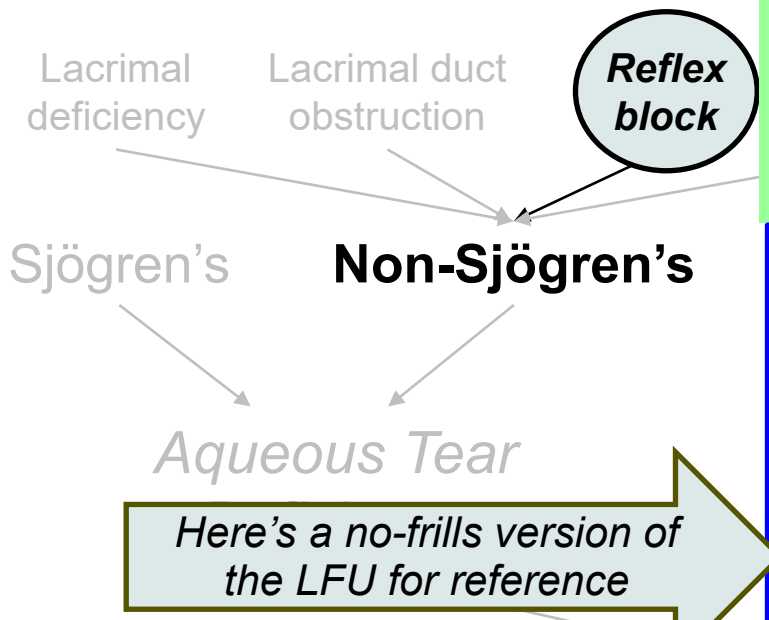
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hypero



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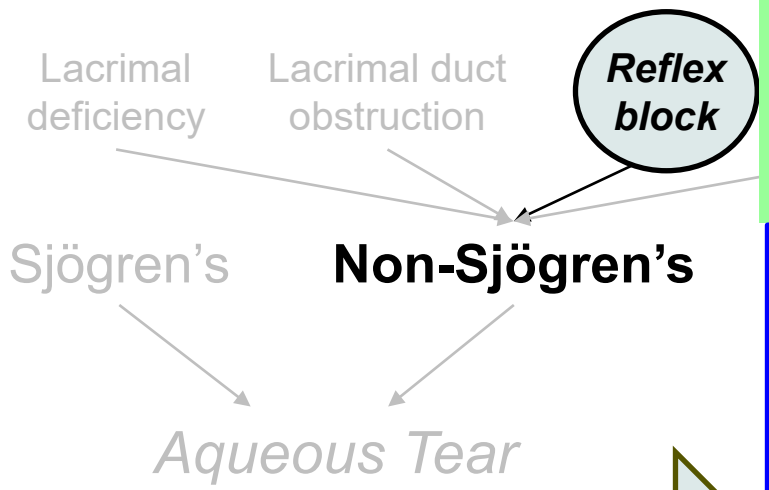
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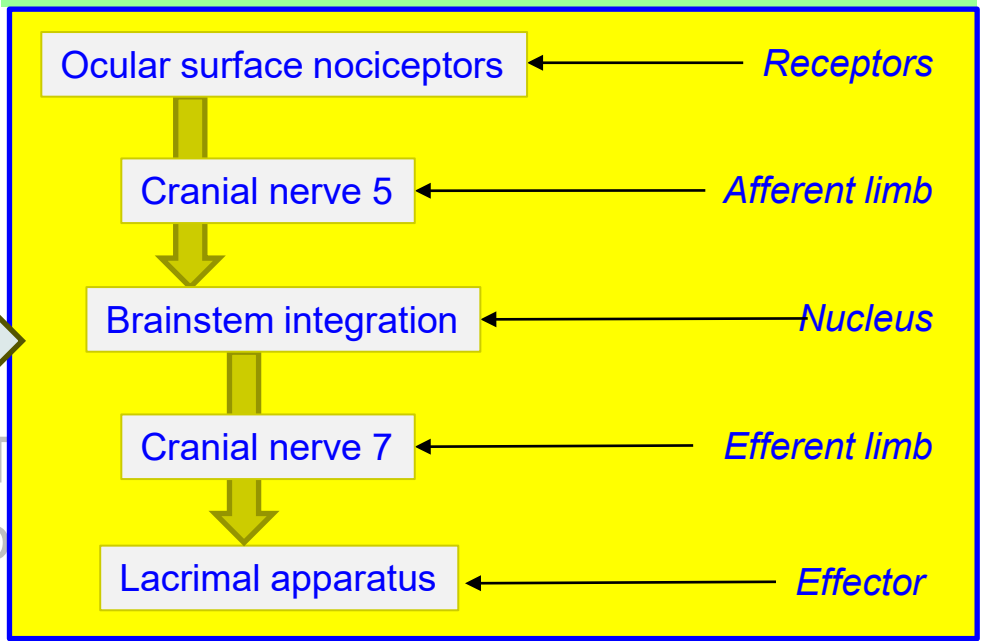
*What are some of the common mechanisms producing efferent limb block?*

- Anything that compromises CN7

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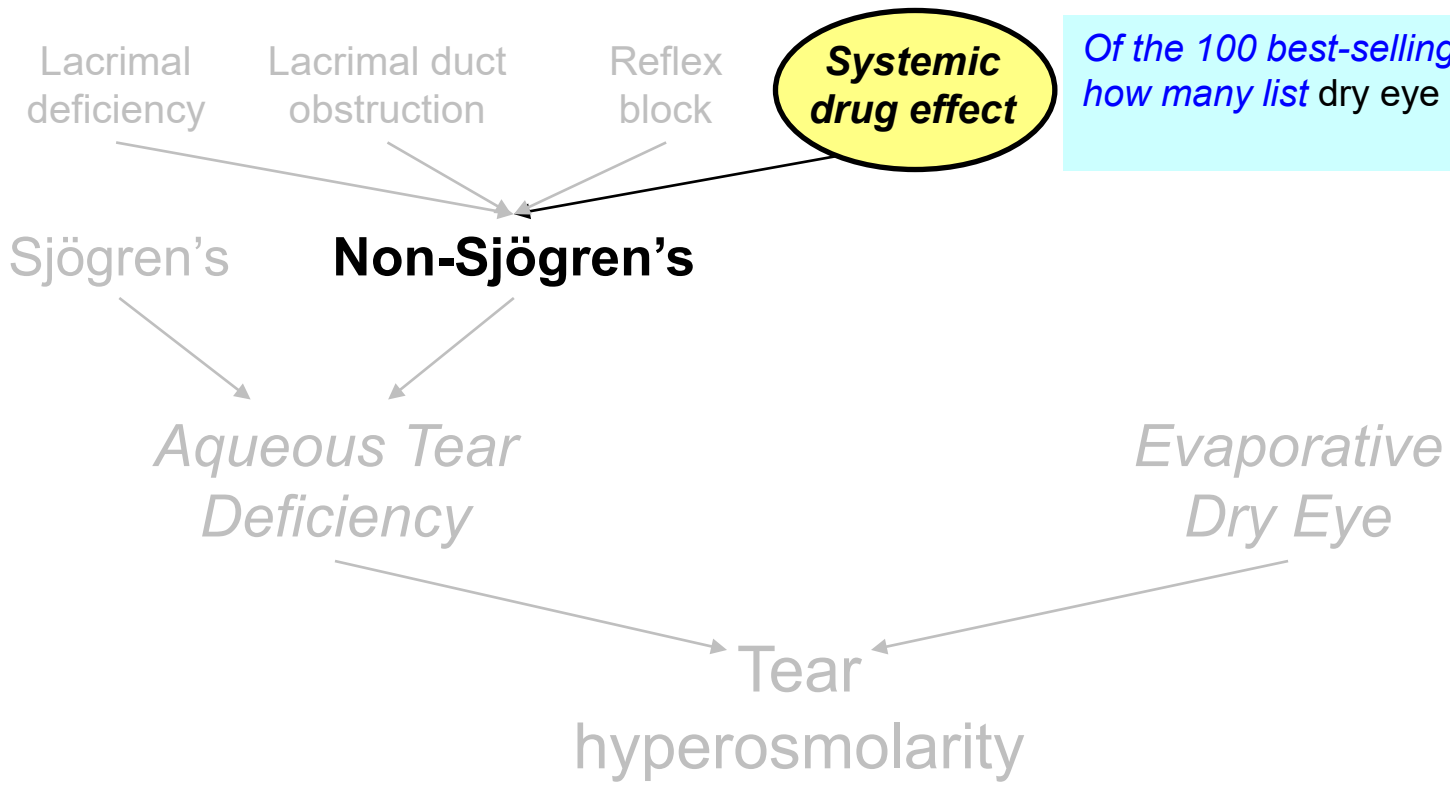
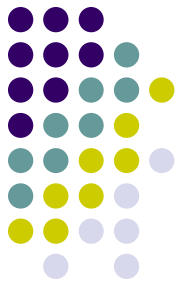


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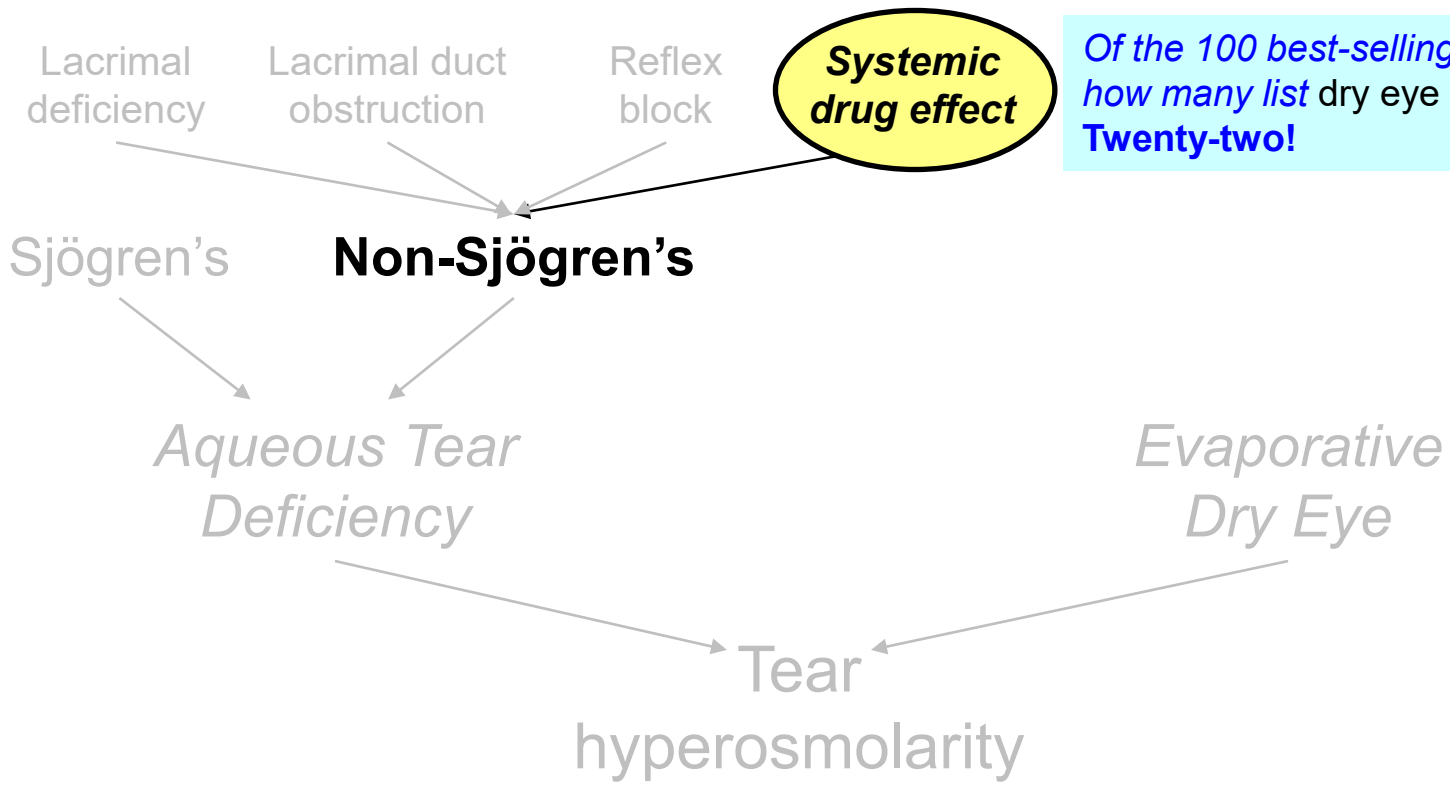
hypero

# Dry Eye Syndrome



*Of the 100 best-selling drugs in the US, how many list dry eye as a side effect?*

# Dry Eye Syndrome



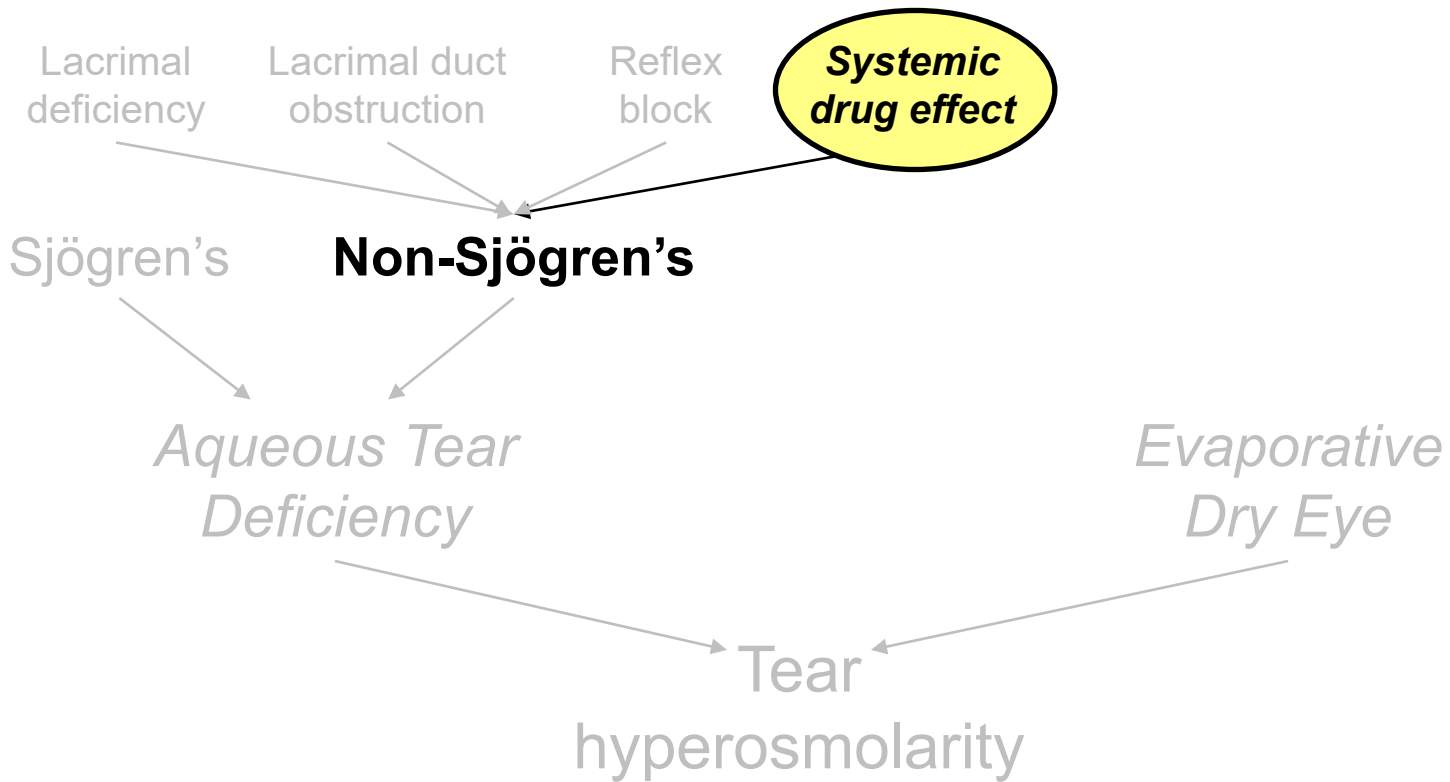
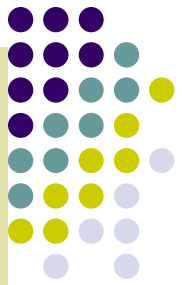
Of the 100 best-selling drugs in the US, how many list dry eye as a side effect? **Twenty-two!**

Three very general classes of pharmacologic effect are implicated in inducing DES—what are they?

Anti- effect

Anti- effect

effect

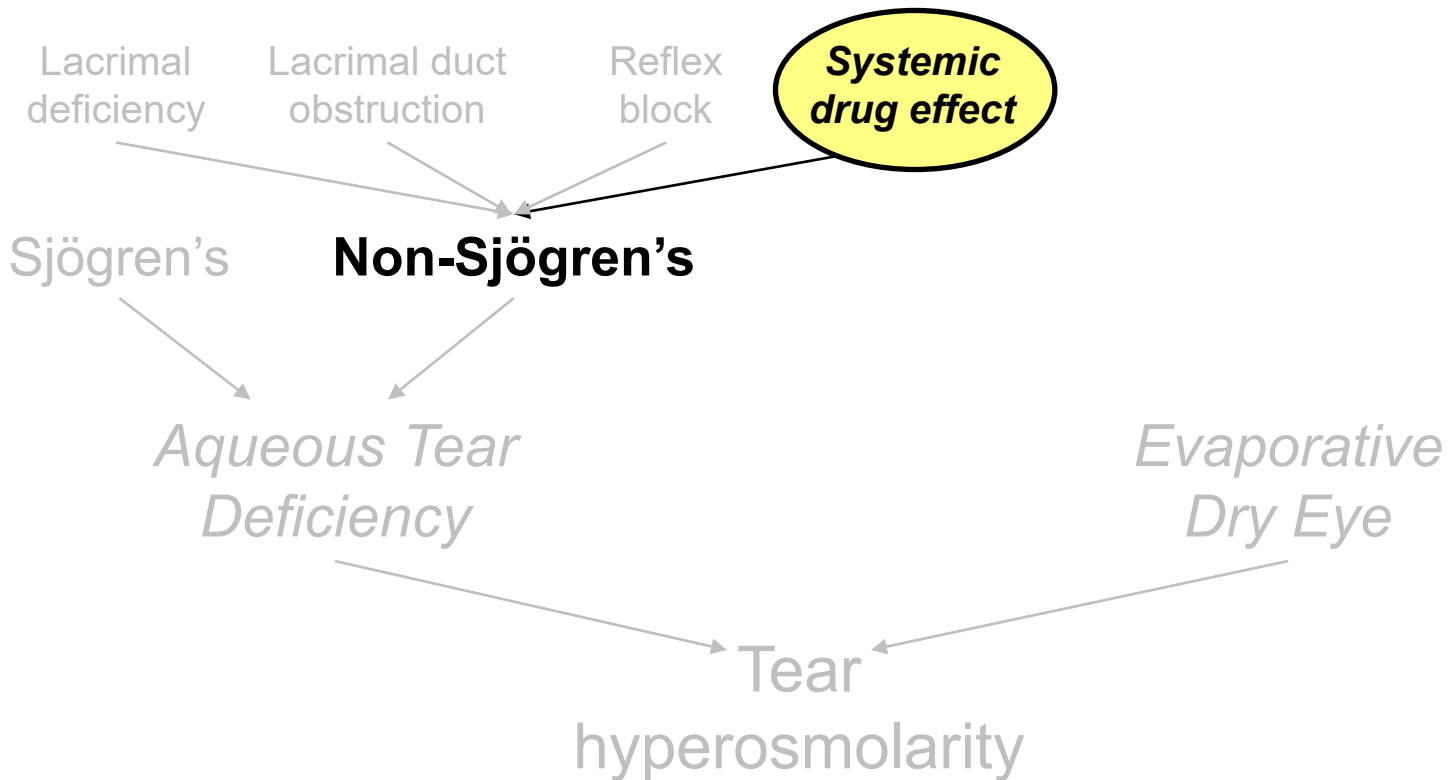
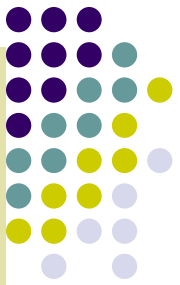


Three very general classes of pharmacologic effect are implicated in inducing DES—what are they?

Anti-histamine effect

Anti-cholinergic effect

Hormonal effect

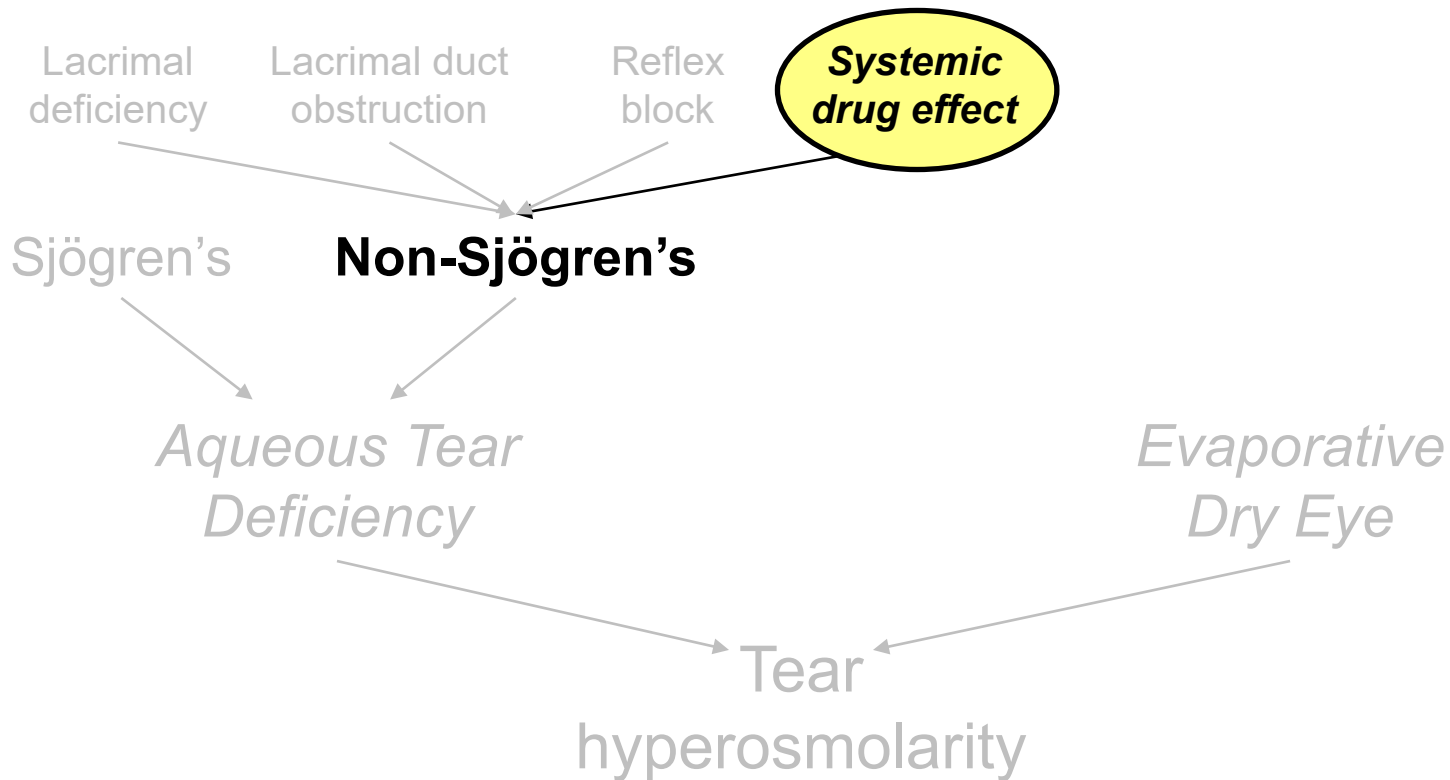
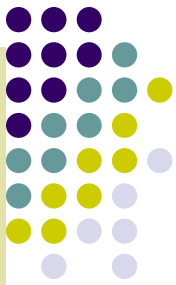


Three very general classes of pharmacologic effect are implicated in inducing DES—what are they?  
What classes of meds are found within each of these effect-groupings?

--? } Anti-histamine effect  
--?

Anti-cholinergic effect

Hormonal effect

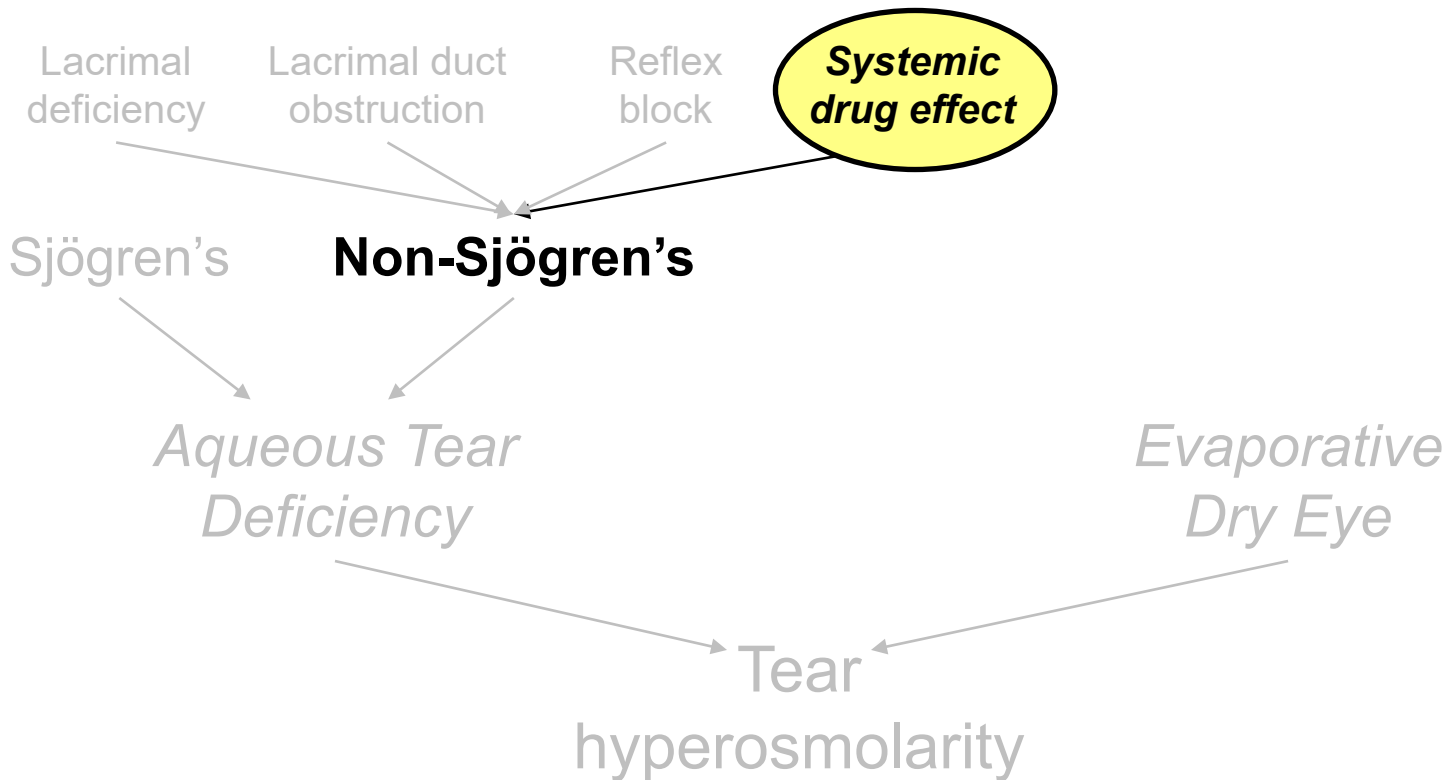
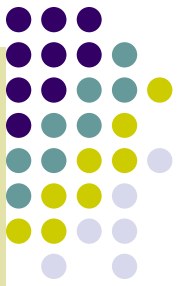


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--Anti-histamines (duh) } Anti-histamine effect  
--Anti-depressants }

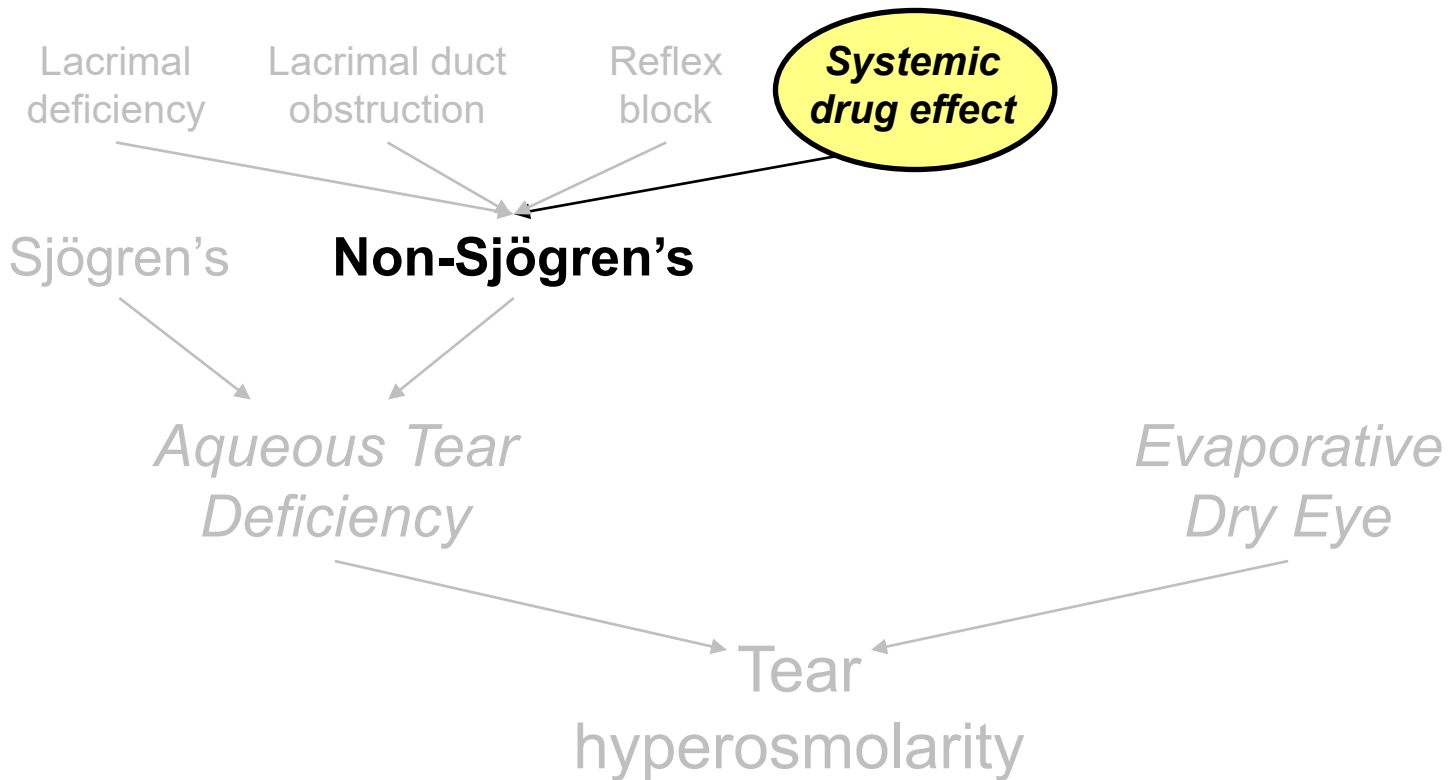
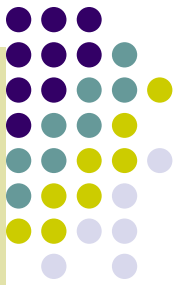
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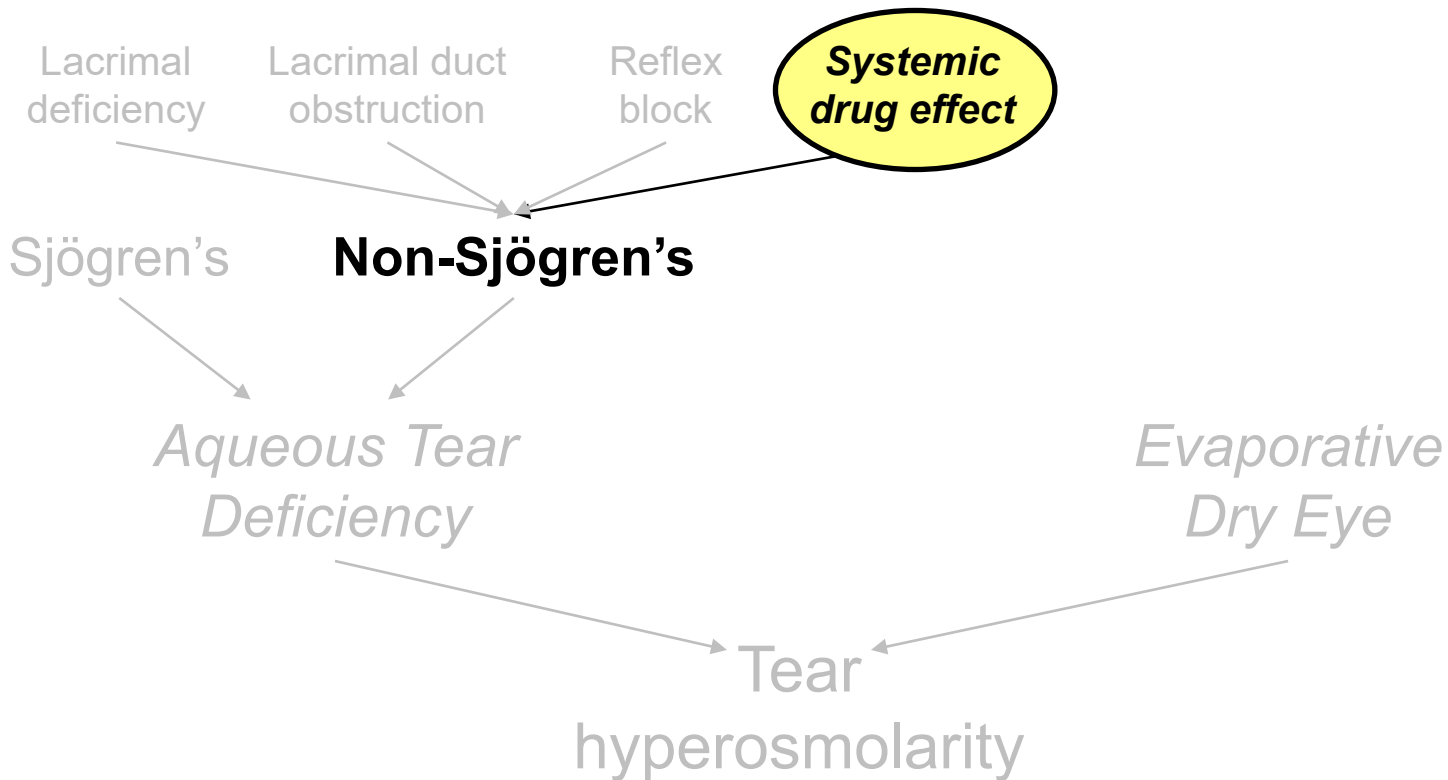
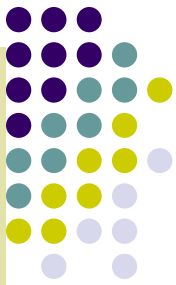
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  - ?
  - ?
  - ?
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- Anti-cholinergic effect
- Hormonal effect





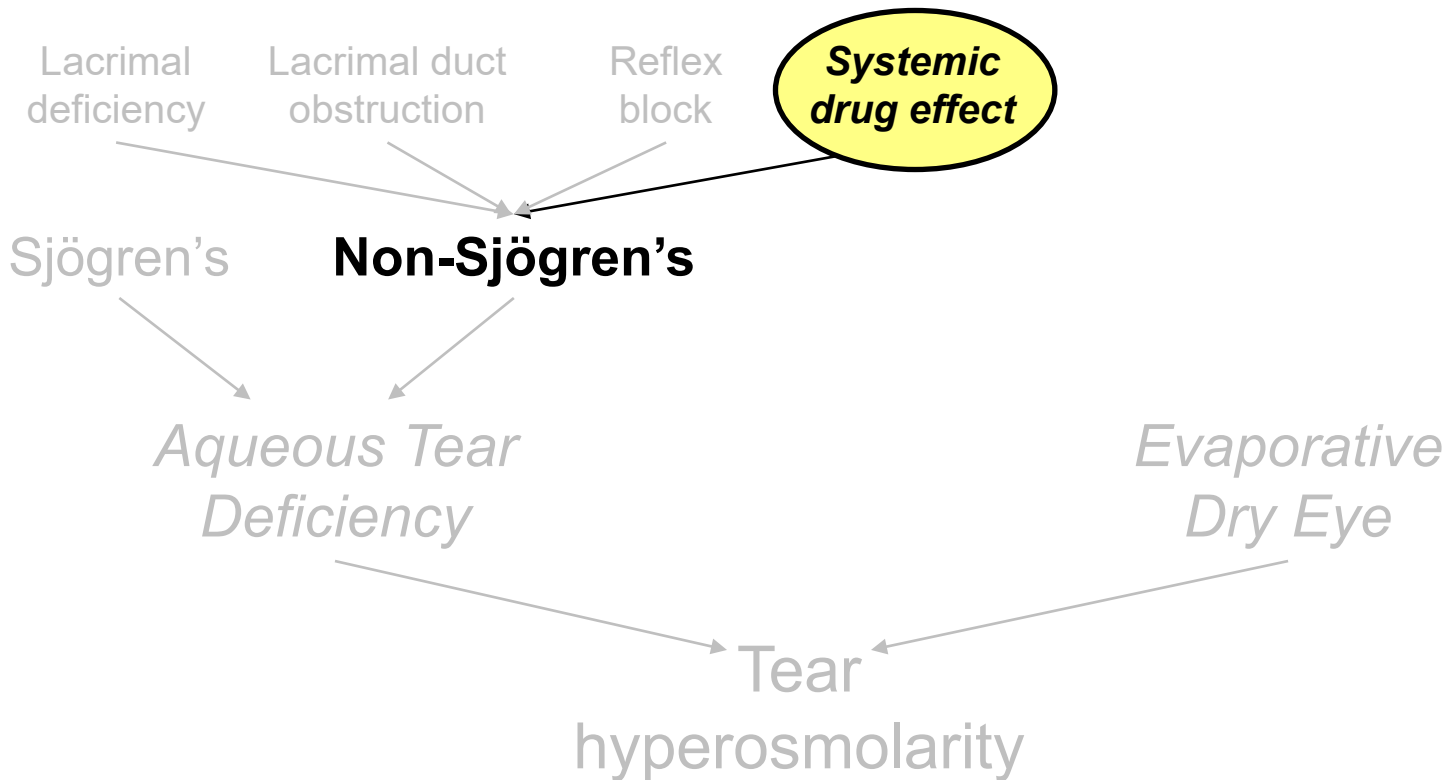
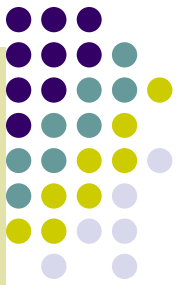
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  - Anti-hypertensives
  - Anti-emetics
  - Anti-Parkinson's
  - Anti-psychotics
- Anti-histamine effect
- Anti-cholinergic effect
- Hormonal effect



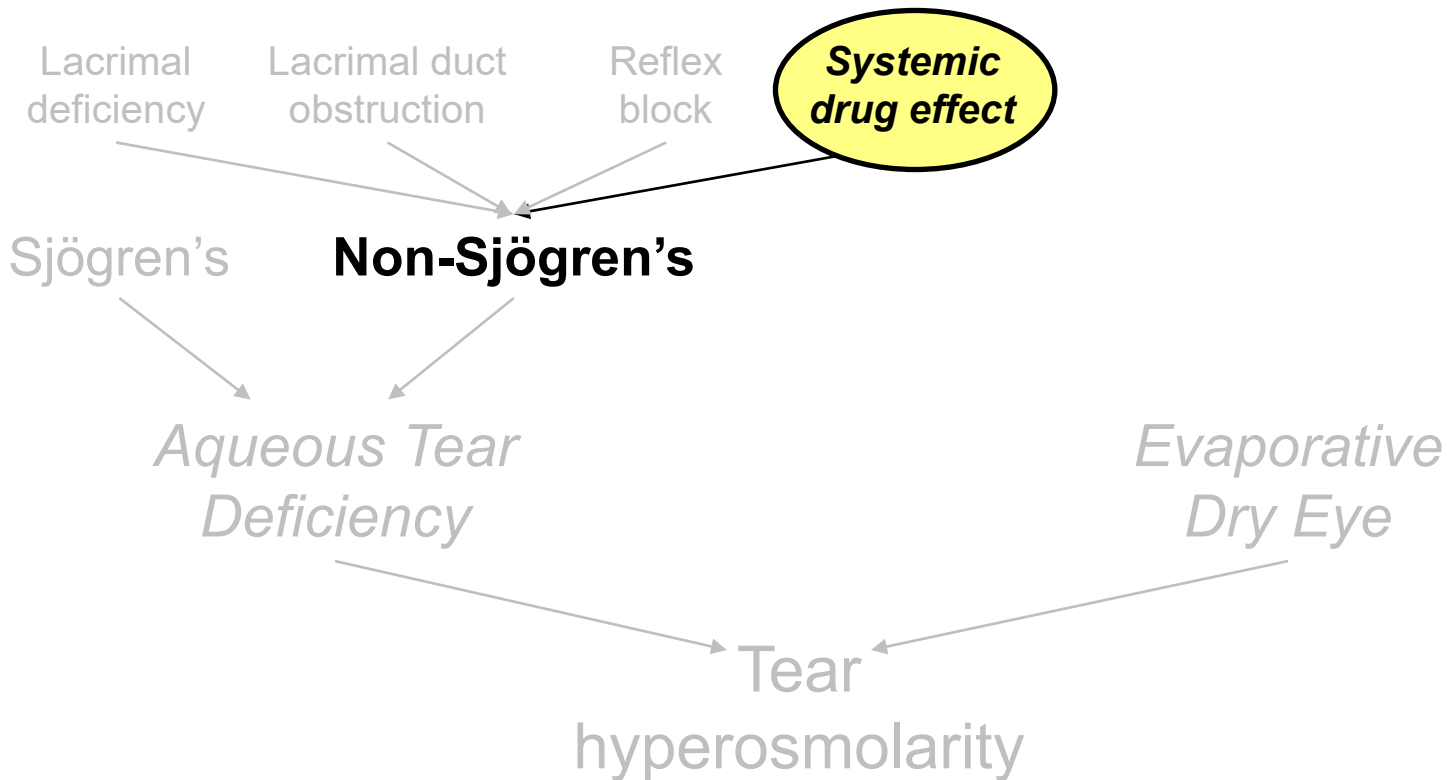
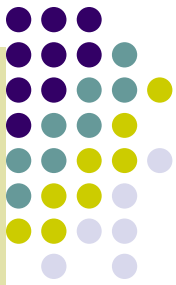
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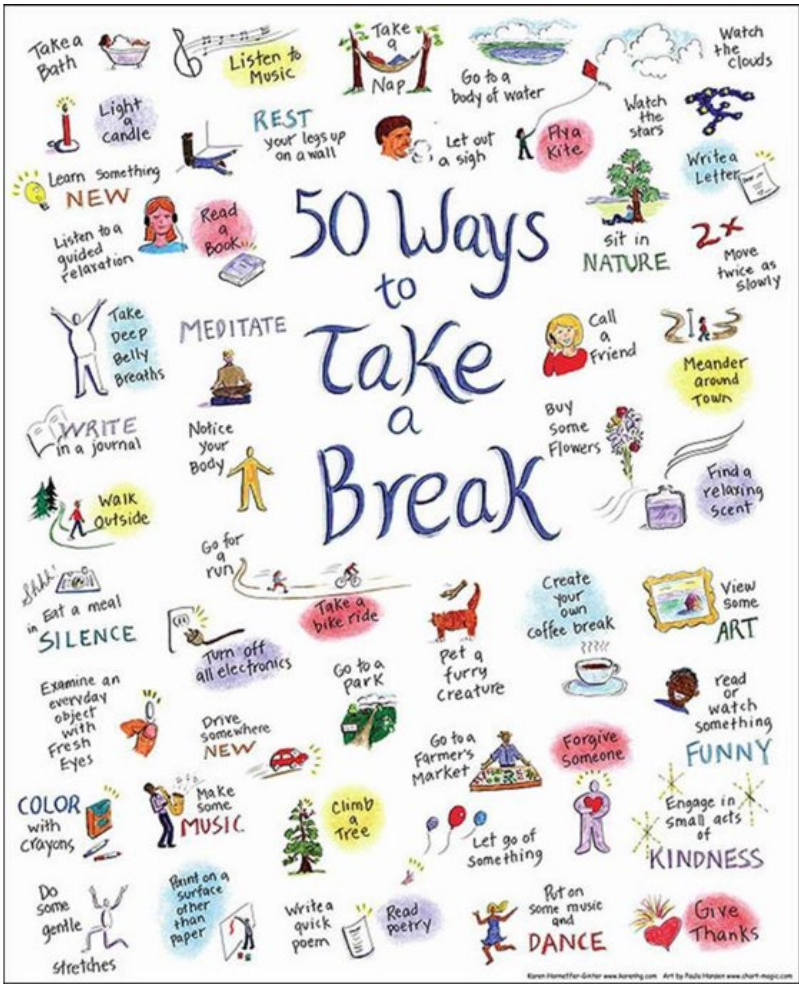


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  - Anti-psychotics
  - Anti-adrenergics
  - Oral contraceptive pills
- Anti-histamine effect
- Anti-cholinergic effect
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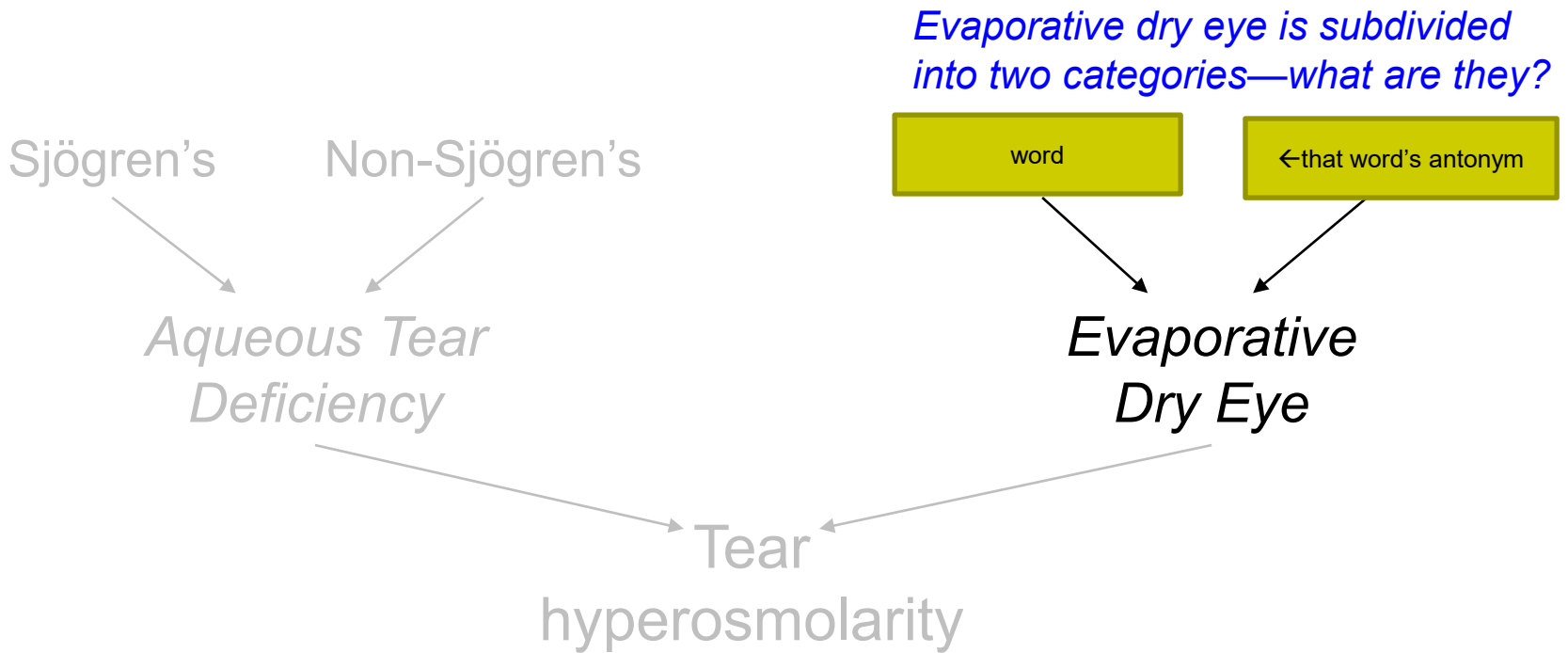


# Dry Eye Syndrome



(This is a good point in the set to take a break)

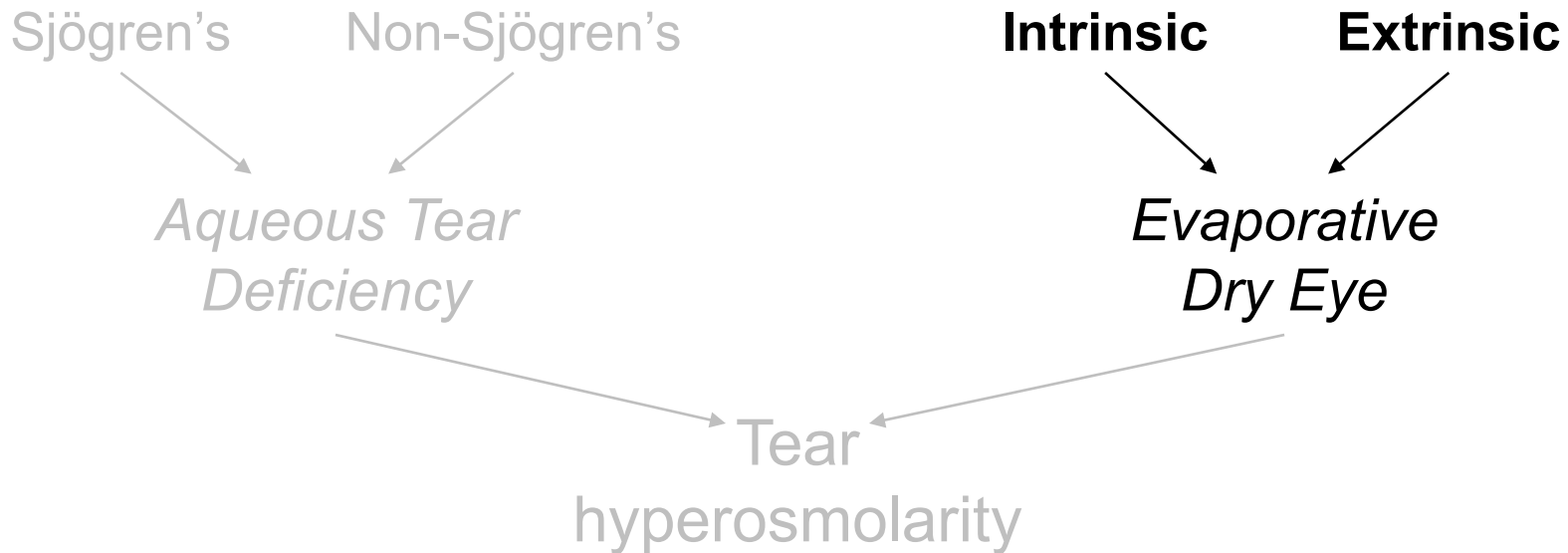
# Dry Eye Syndrome



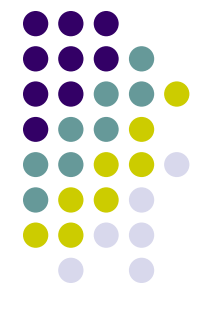
# Dry Eye Syndrome



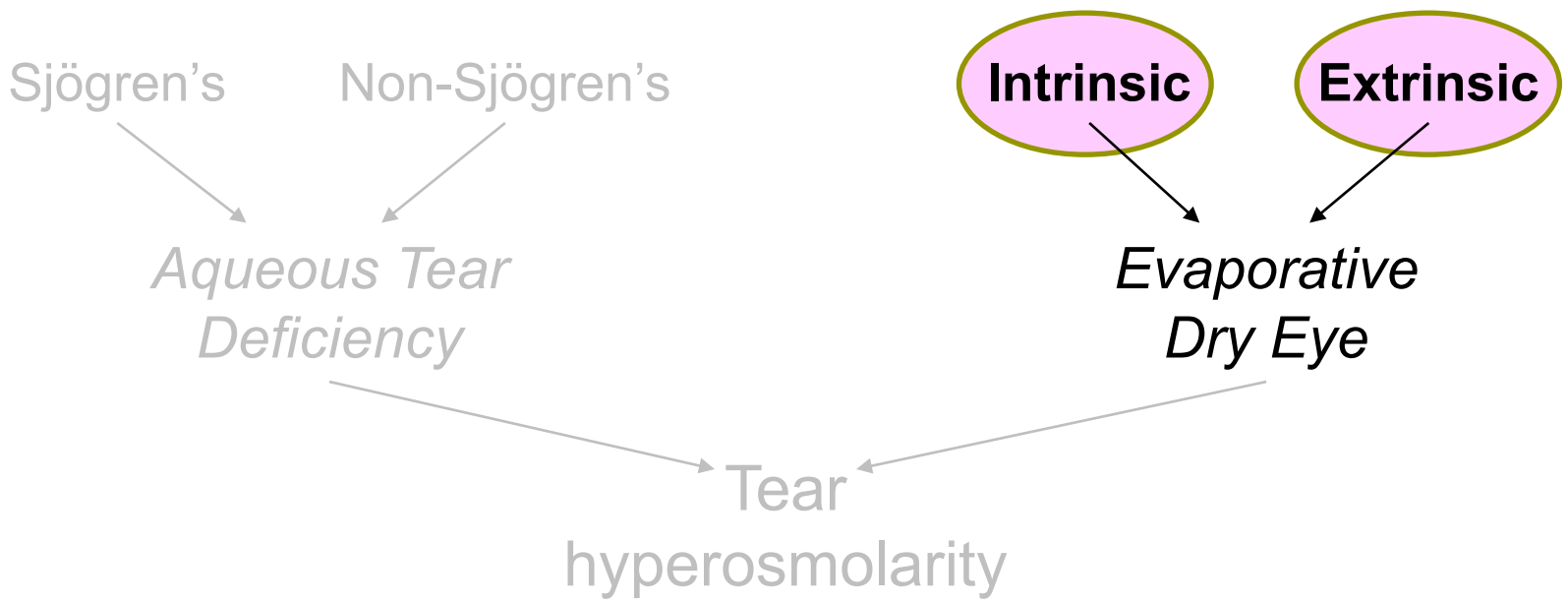
*Evaporative dry eye is subdivided into two categories—what are they?*



# Dry Eye Syndrome



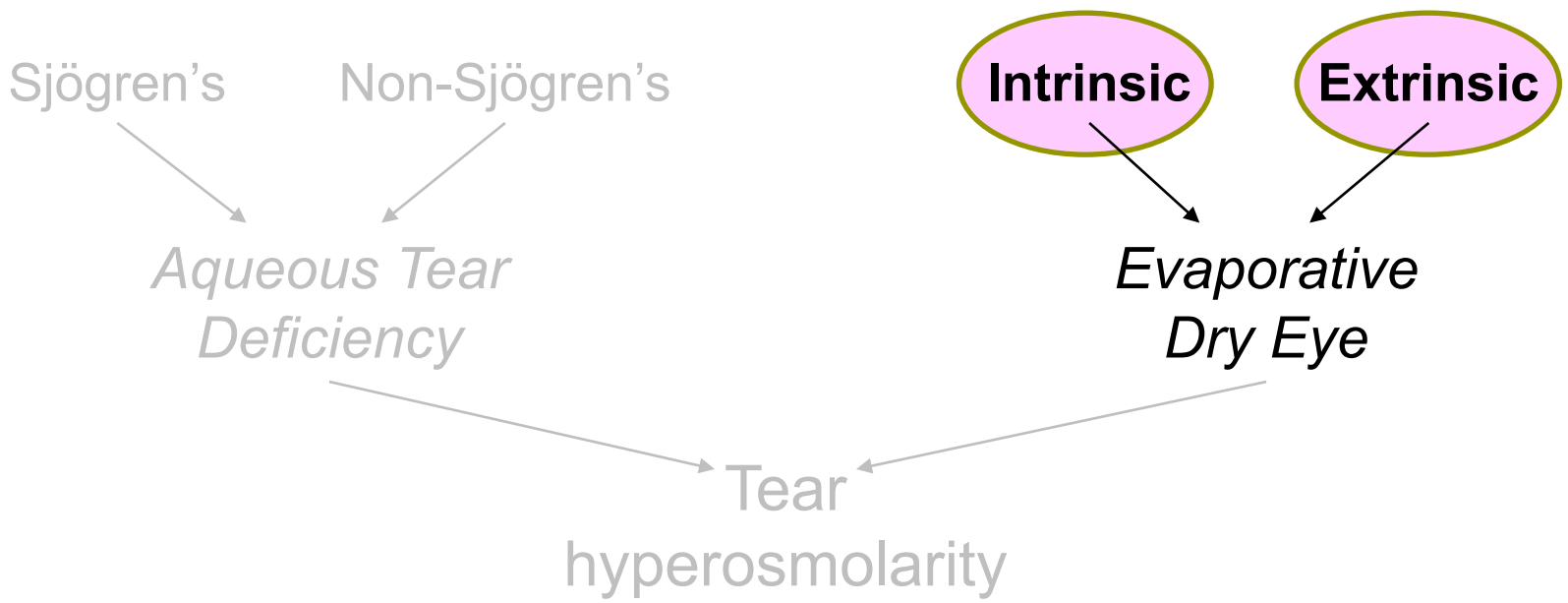
*In this context, to what do the terms intrinsic and extrinsic refer?*



# Dry Eye Syndrome

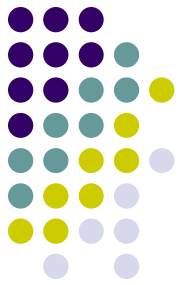


*In this context, to what do the terms intrinsic and extrinsic refer?*  
*Intrinsic* evaporative dry eye refers to any cause related to the eyelids.  
*Extrinsic* refers to any non-eyelid factor that promoted evaporation.



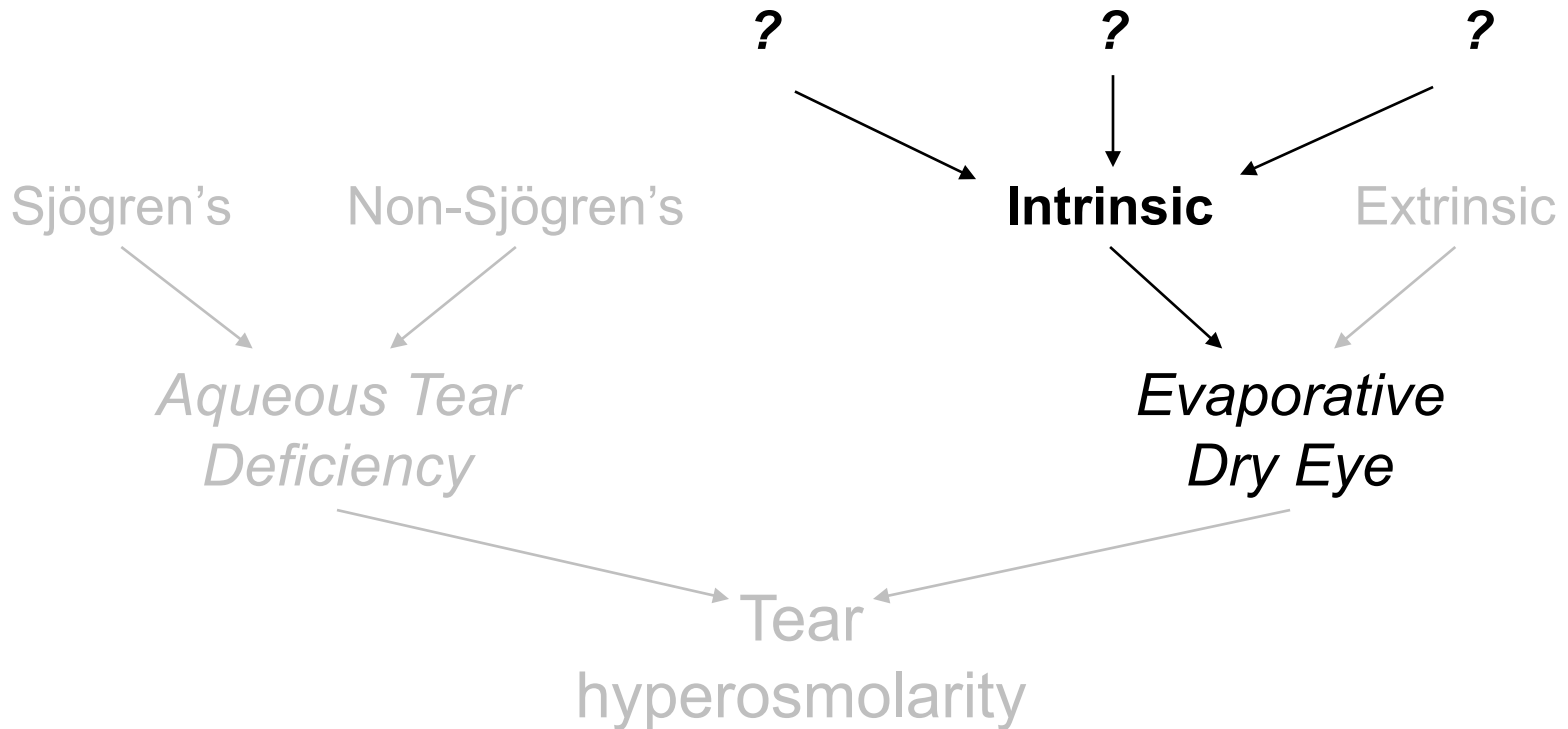


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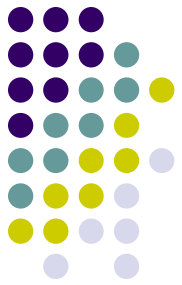


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*What are the three main etiologies of intrinsic evaporative dry eye?*

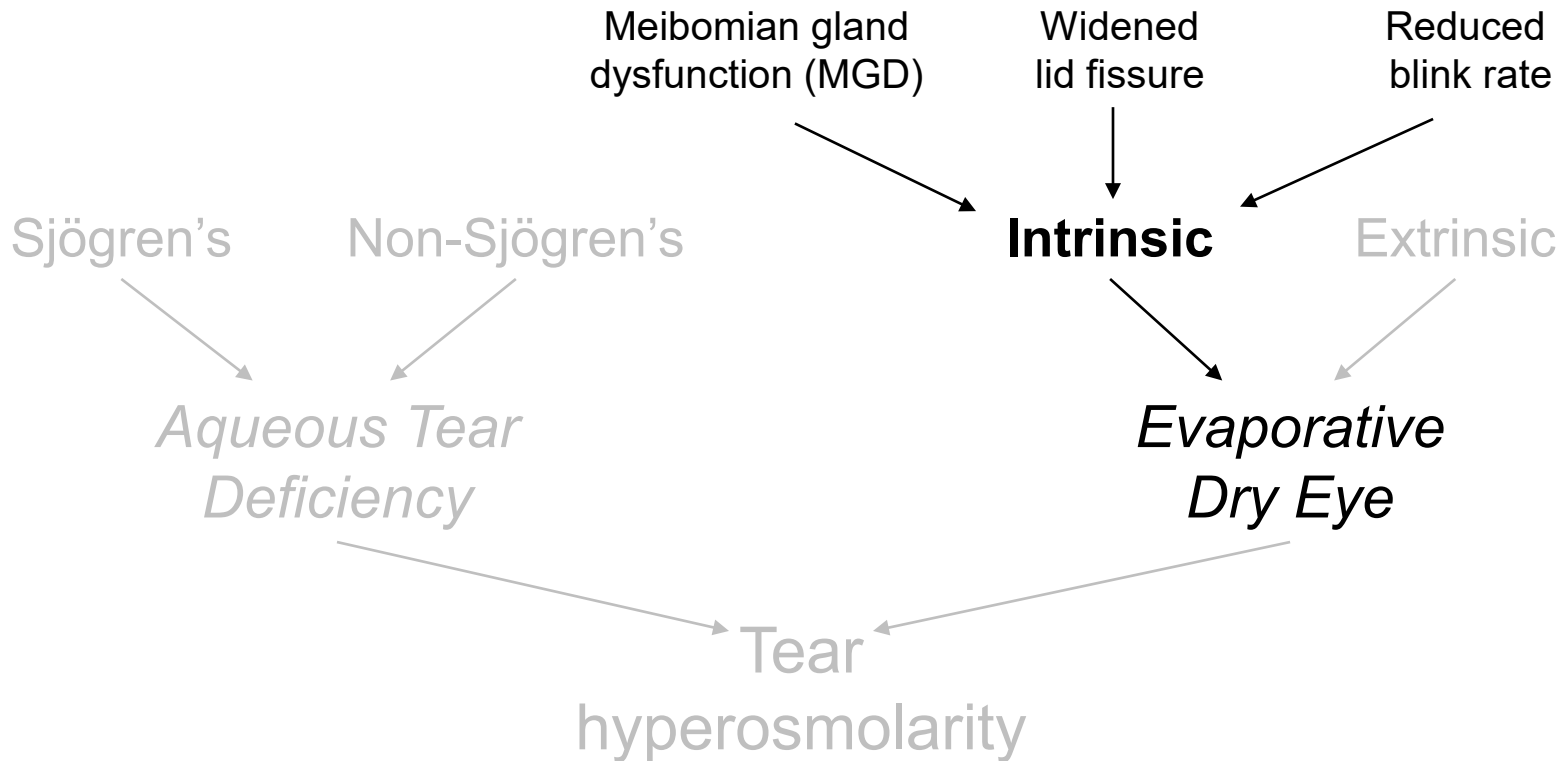


# Dry Eye Syndrome



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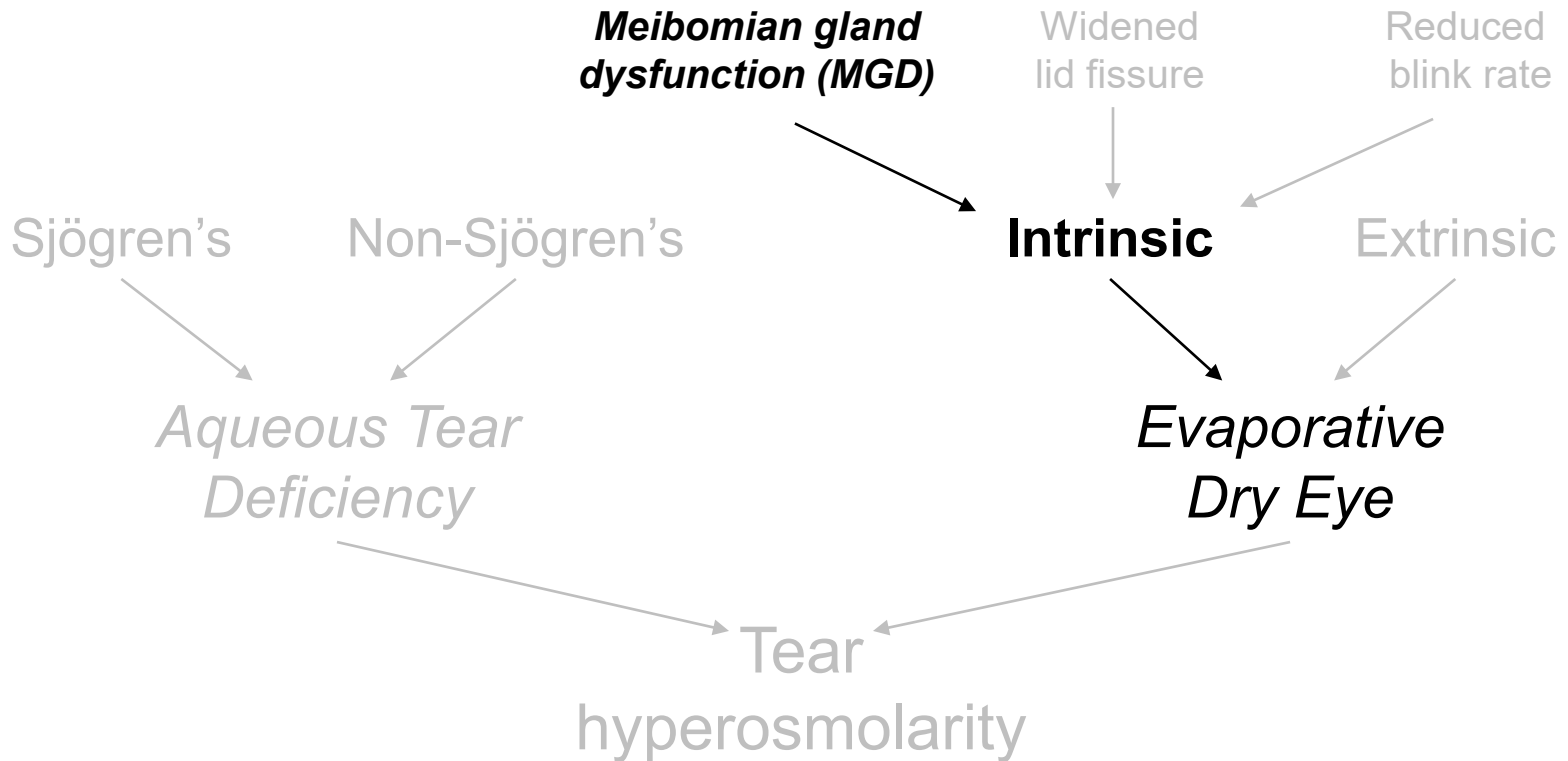
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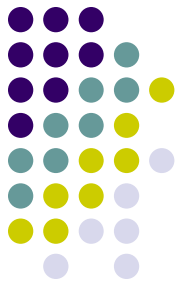
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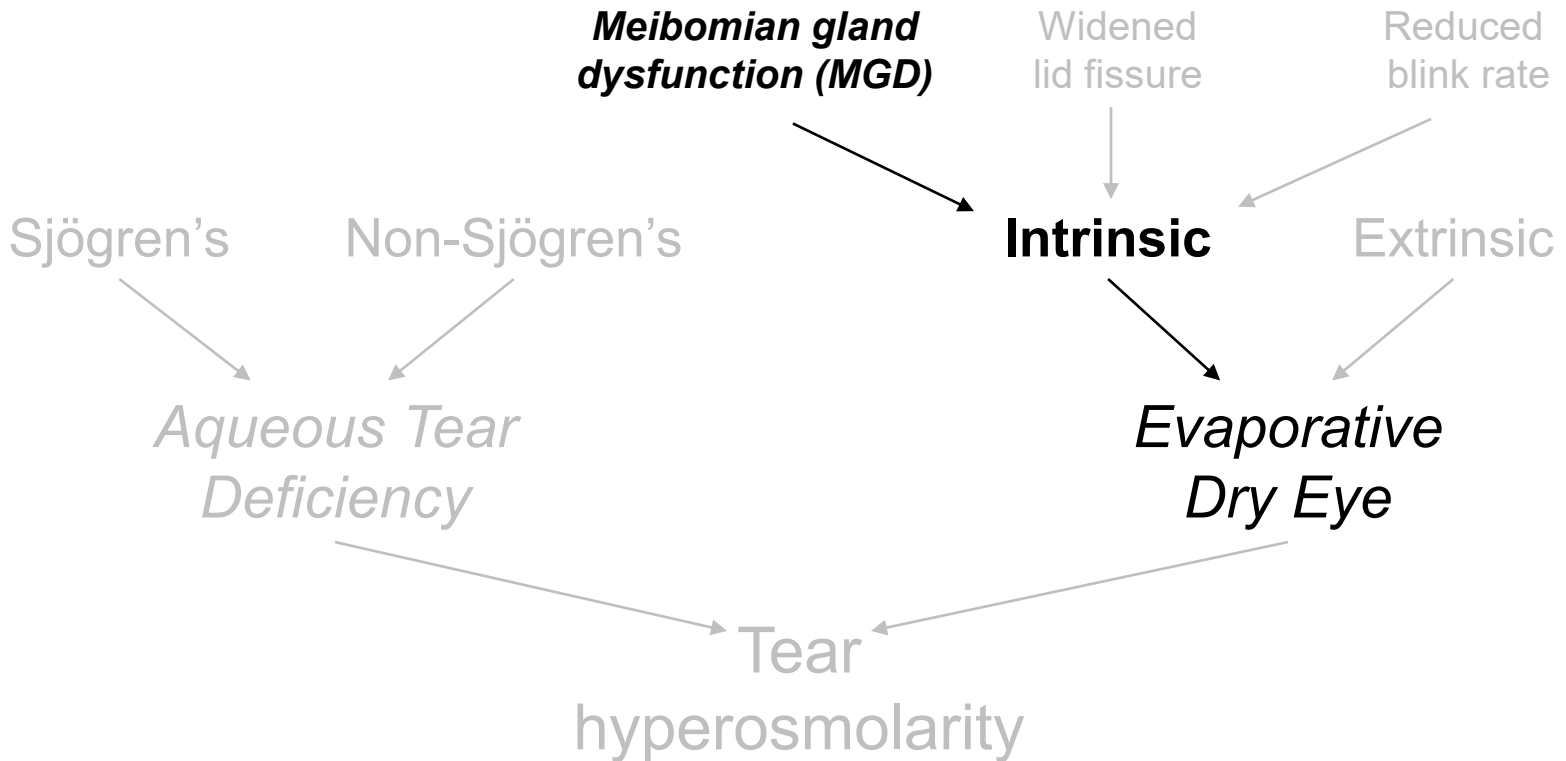
*MGD demonstrates a racial predilection—what group has a notably higher prevalence?*



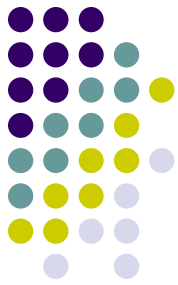
# Dry Eye Syndrome



*MGD demonstrates a racial predilection—what group has a notably higher prevalence?  
Asians*

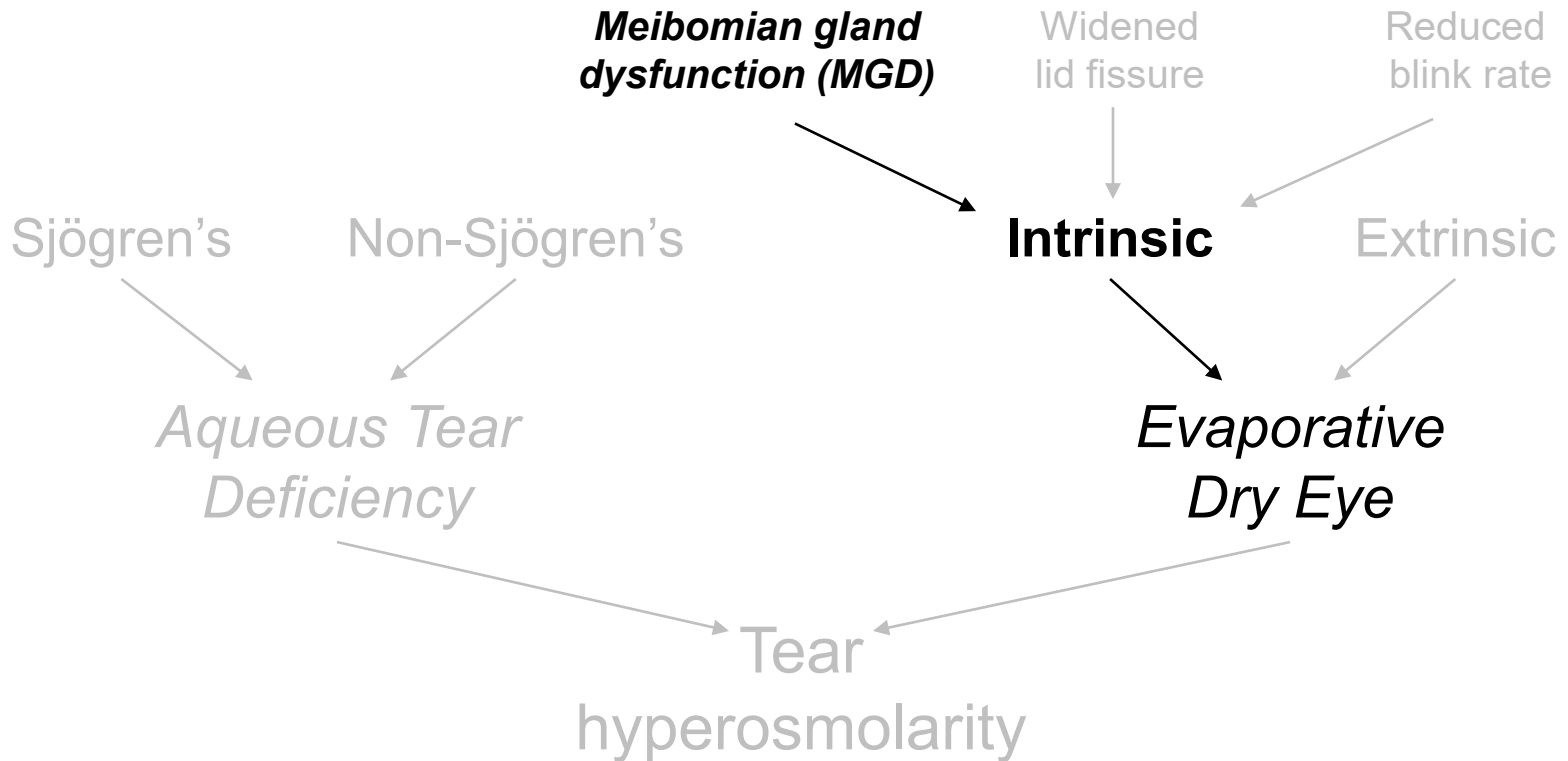


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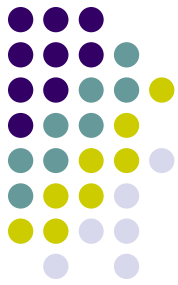


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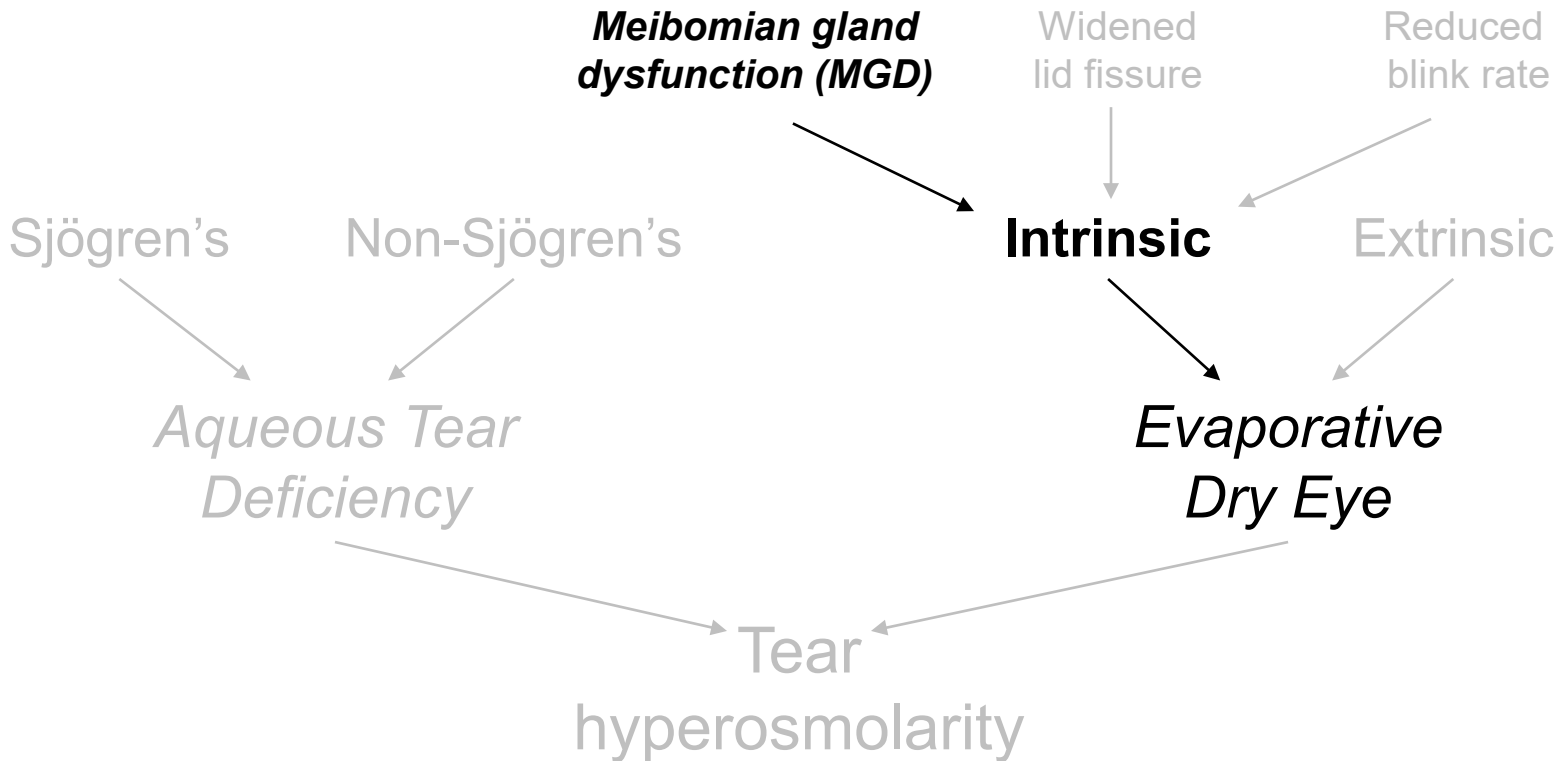
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**Reduction** of gland output leading to inadequate volume of tear-film meibum

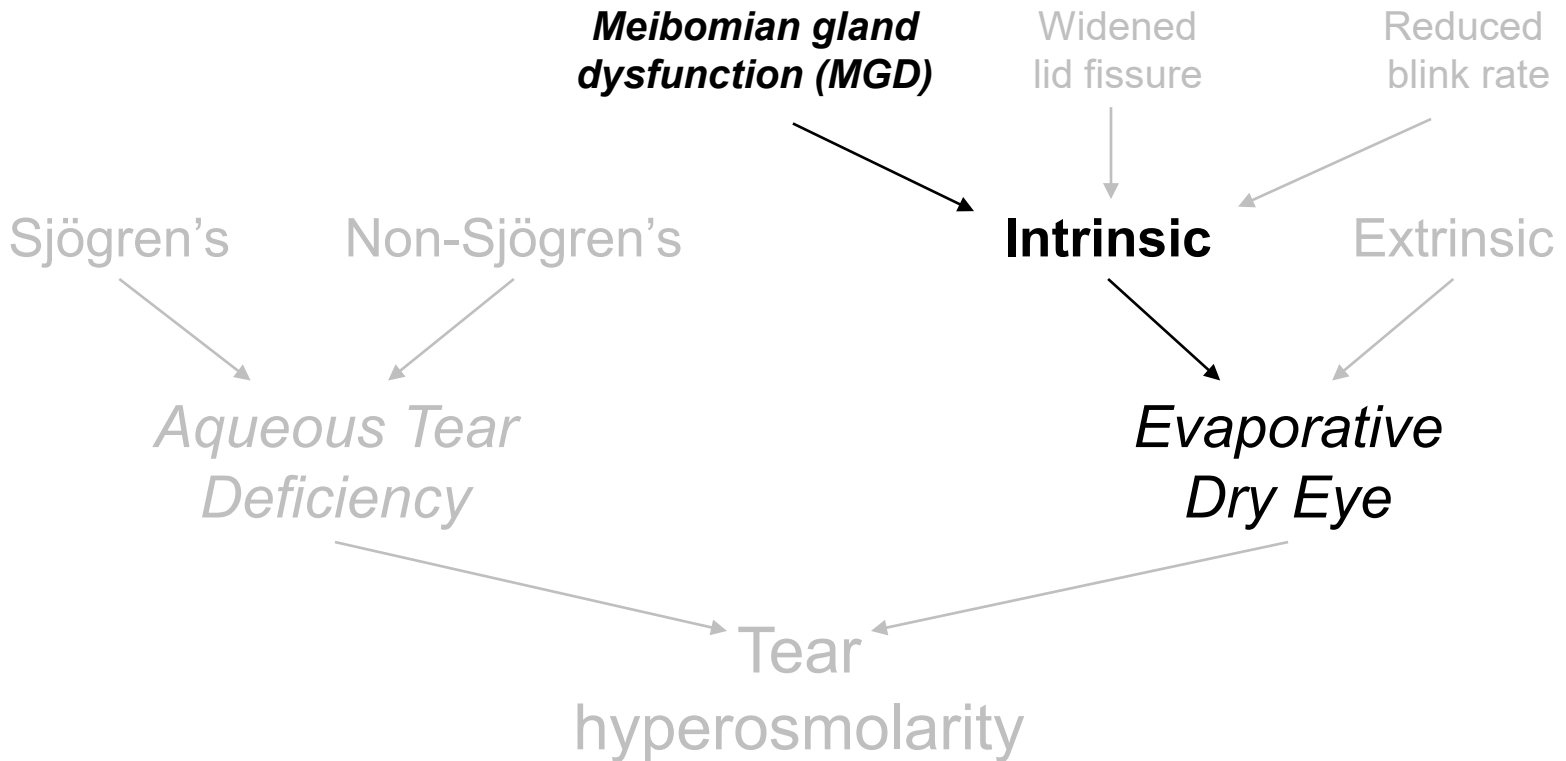


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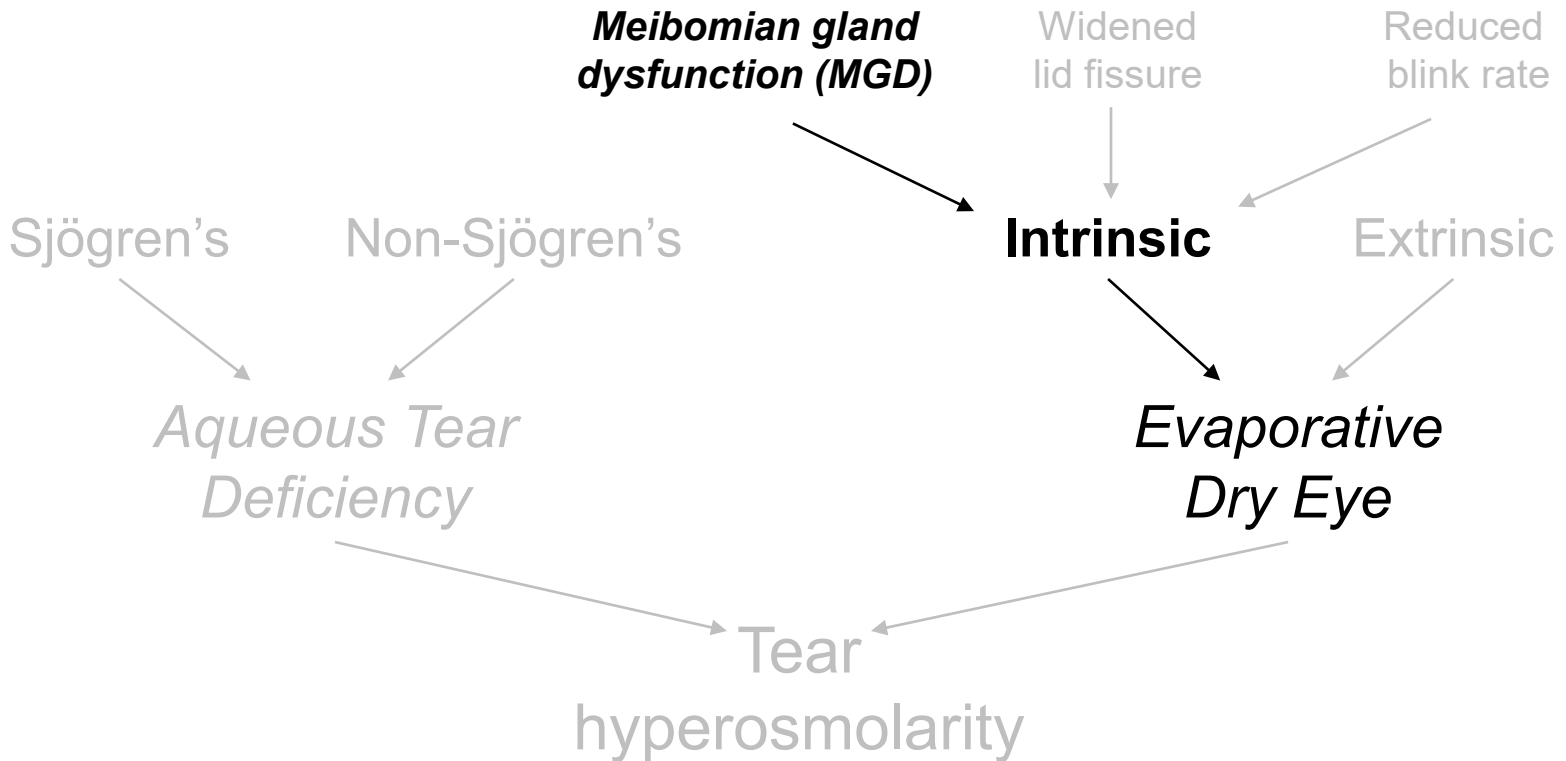
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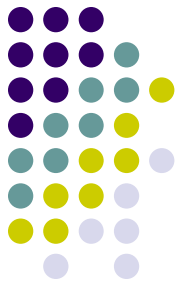
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*Obstructive MGD is divided into two subtypes—what are they?*





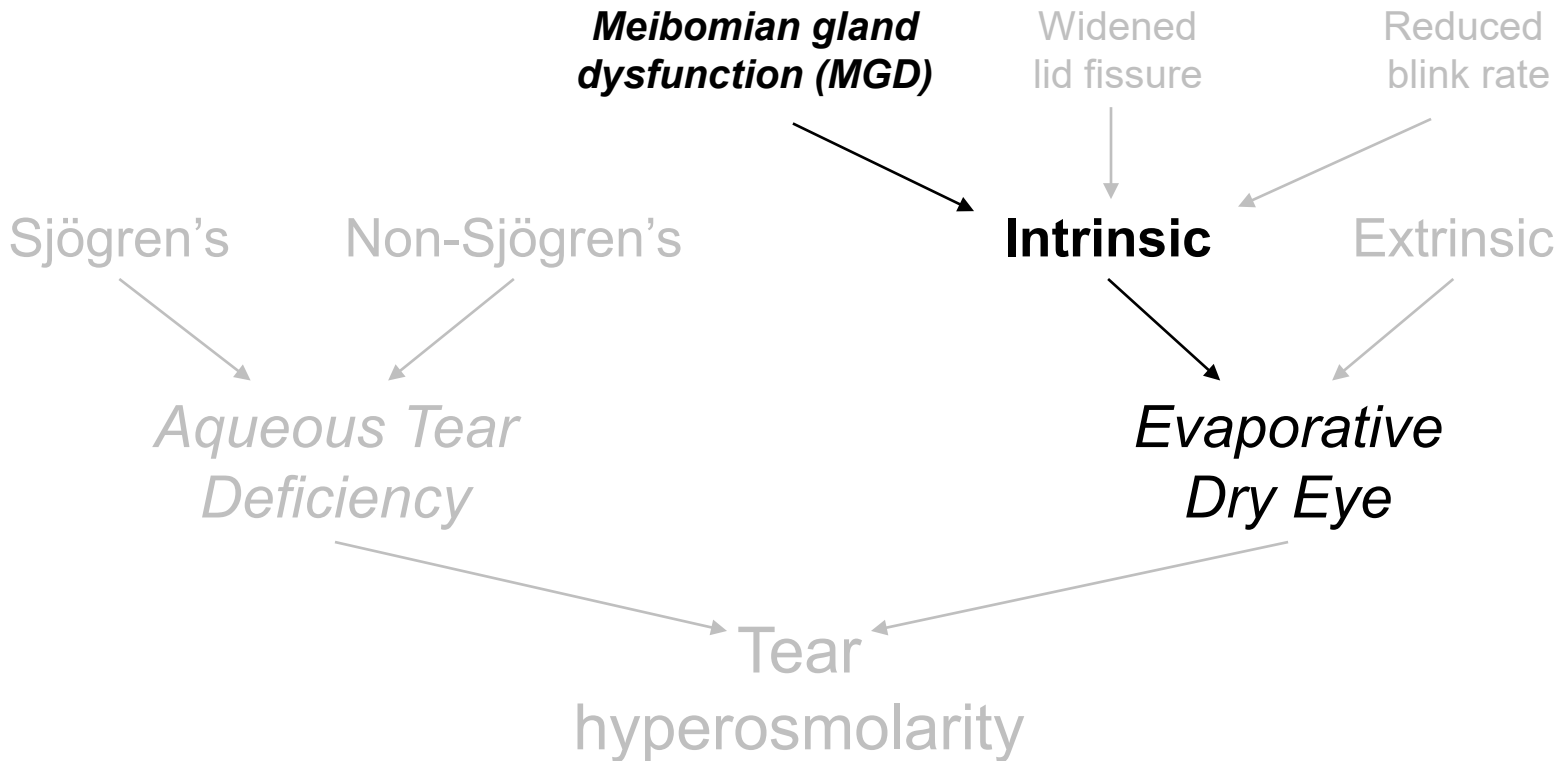
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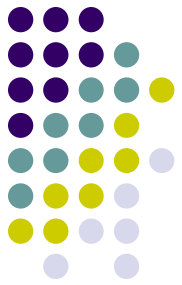
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Obstructive MGD is divided into two subtypes—what are they?  
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**Meibomian gland**

Widened

Reduced  
blink rate

The Cornea book highlights three causes of cicatrizing obstructive MGD—  
what are they?

--?

--?

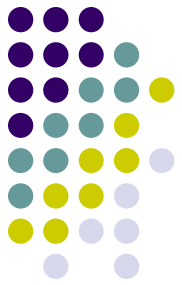
--?

**C** Extrinsic

Evaporative  
Dry Eye

Tear  
hyposmolarity

# Dry Eye Syndrome



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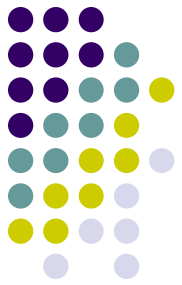
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**C**      Extrinsic  
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what are they?

--Trachoma

--Mucous-membrane pemphigoid (aka three words)

--Atopy

**c**

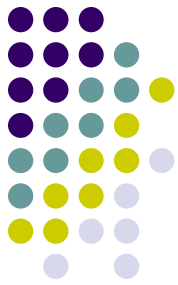
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- Atopy

**C**      Extrinsic  
Evaporative  
Dry Eye

Tear  
hyposmolarity

# Dry Eye Syndrome



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## Meibomian gland

Widened

Reduced  
blink rate

The Cornea book highlights three causes of cicatrizing obstructive MGD—  
what are they?

- Trachoma
- Mucous-membrane pemphigoid (aka *ocular cicatricial pemphigoid*)
- Atopy

The book highlights three causes of **noncicatrizing** obstructive MGD—  
what are **they**?

- ?
- ?
- ?

**C** Extrinsic  
Evaporative  
Dry Eye

Tear  
hyposmolarity

# Dry Eye Syndrome



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## Meibomian gland

Widened

Reduced  
blink rate

The Cornea book highlights three causes of cicatrizing obstructive MGD—  
what are they?

- Trachoma
- Mucous-membrane pemphigoid (aka *ocular cicatricial pemphigoid*)
- Atopy

The book highlights three causes of **noncicatrizing** obstructive MGD—  
what are **they**?

- Rosacea
- Seborrheic dermatitis
- Atopy

**C** Extrinsic  
Evaporative  
Dry Eye

Tear  
hyposmolarity

# Dry Eye Syndrome



MGD demonstrates a racial predilection—what group has a notably higher prevalence?  
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- Trachoma
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The book highlights three causes of **noncicatrizing** obstructive MGD—what are they?

- Rosacea
- Seborrheic dermatitis
- Atopy**

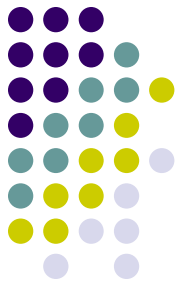
Not a typo (on my part)—the Cornea book lists atopy under **both** causes

**C** Extrinsic  
Evaporative  
Dry Eye

Tear  
hyposmolarity



# Dry Eye Syndrome



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Widened

Reduced  
blink rate

*In a nutshell, what is rosacea?*

The Co  
what ar  
--Trache  
--Mucos  
--Atopy

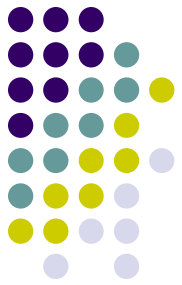
The bo  
what ar  
--Rosacea  
--Sebor  
--Atopy

Extrinsic

orative  
Eye

**Rosacea**

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Widened

Reduced  
blink rate

*In a nutshell, what is rosacea?*

A acute vs chronic skin condition often involving the eyelids

Extrinsic

orative  
Eye

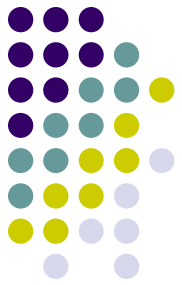
The Co  
what ar  
--Trach  
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--Atopy

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**Rosacea**



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Widened

Reduced  
blink rate

*In a nutshell, what is rosacea?*

A chronic skin condition often involving the eyelids

Extrinsic

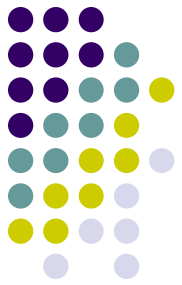
orative  
Eye

The Co  
what ar  
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--Mucos  
--Atopy

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**Rosacea**

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*Meibomian gland*

Widened

Reduced  
blink rate

*In a nutshell, what is rosacea?*

A chronic skin condition often involving the eyelids

*What is the cause?*

Extrinsic

orative  
Eye

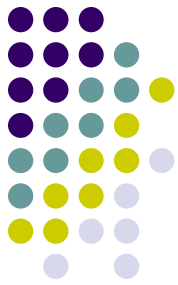
The Co  
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**Rosacea**



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Widened

Reduced  
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*What is the cause?*

It is unknown at this time

Extrinsic

orative  
Eye

The Co  
what ar  
--Trach  
--Mucoc  
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A chronic skin condition often involving the eyelids

*What is the cause?*  
It is unknown at this time

*Is there a gender predilection?*

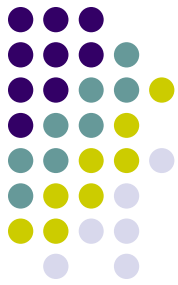


The bo  
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*What is the cause?*

It is unknown at this time

*Is there a gender predilection?*

Yes **M vs F** are more likely to be affected

Extrinsic

orative  
Eye

The Co  
what ar  
--Trach  
--Mucoc  
--Atopy  
  
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Extrinsic

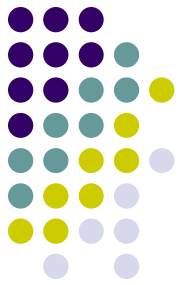
or  
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what ar  
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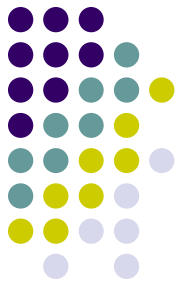
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It is unknown at this time

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Yes, ♀ are more likely to be affected. No.

The bo  
what ar  
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Extrinsic

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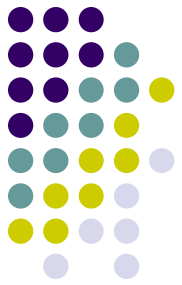
*Is there a gender predilection? A racial predilection? Age predilection?*  
Yes, ♀ are more likely to be affected. No.

The bo  
what ar  
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A chronic skin condition often involving the eyelids

*What is the cause?*

It is unknown at this time

*Is there a gender predilection? A racial predilection? Age predilection?*

Yes, ♀ are more likely to be affected. No. Middle-aged.

Extrinsic

orative  
Eye

The Co  
what ar  
--Trach  
--Mucoc  
--Atopy  
  
The bo  
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--Rosacea  
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A chronic skin condition often involving the eyelids

What is the cause?  
It is unknown at this time

Is there a gender predilection? A racial predilection? Age predilection?  
Yes, ♀ are more likely to be affected. N **Tender-aged?**

Extrinsic

The bo  
what ar  
**Rosacea**  
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--Atopy

Can young individuals get rosacea?

# Dry Eye Syndrome



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Yes, ♀ are more likely to be affected. N **Tender-aged?**

Extrinsic

The bo  
what ar  
**Rosacea**  
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--Atopy

Can young individuals get rosacea?  
They can indeed

# Dry Eye Syndrome



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*What is the cause?*  
It is unknown at this time

*Is there a gender predilection? A racial predilection? Age predilection?*  
Yes, ♀ are more likely to be affected. N

**Tender-aged?**

Extrinsic

The bo  
what ar  
--Rosacea  
--Sebor  
--Atopy

**Rosacea**

Can young individuals get rosacea?  
They can indeed

What is the classic tipoff that a **young** person has rosacea?

# Dry Eye Syndrome



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Yes, ♀ are more likely to be affected. N **Tender-aged?**

Extrinsic

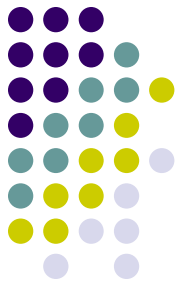
The bo  
what ar  
**Rosacea**  
--Sebor  
--Atopy

Can young individuals get rosacea?  
They can indeed

What is the classic tipoff that a young person has rosacea?  
A hx of recurrent



# Dry Eye Syndrome



MGD demonstrates a racial predilection—what group has a notably higher prevalence?

Asians

In one simple word, what is the underlying issue in most cases of MGD?

**Obstruction** of gland output leading to inadequate volume of tear-film meibum

Obstructive MGD is divided into two subtypes—what are they?

**Cicatrizing** and noncicatrizing

**Meibomian gland**

Widened

Reduced  
blink rate

The Co  
what ar  
--Trach  
--Mucoc  
--Atopy

*In a nutshell, what is rosacea?*

A chronic skin condition often involving the eyelids

*What is the cause?*

It is unknown at this time

Extrinsic

The bo  
what ar

*Is there a gender predilection? A racial predilection? Age predilection?*

Yes, ♀ are more likely to be affected. **Tender-aged?**

**Rosacea**

--Sebor  
--Atopy

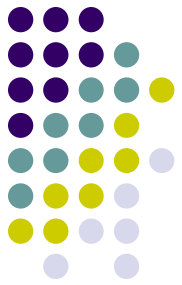
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Extrinsic

orative  
Eye

The Co  
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--Trach  
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The bo  
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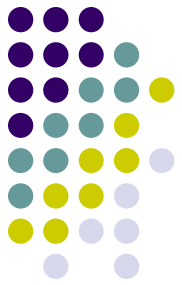
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Eye

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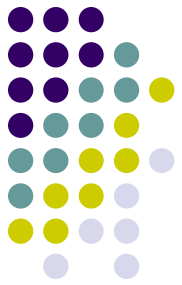
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--Midface

--?

--?

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The bo  
what ar  
--Rosacea  
--Sebor  
--Atopy

**Rosacea**

*What are the classic nonocular findings on exam?*  
--Midface erythema  
--?  
--?

Extrinsic  
orative  
Eye

# Dry Eye Syndrome



Rosacea: Midface erythema

# Dry Eye Syndrome



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Extrinsic

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Eye

The Co  
what ar  
--Trach  
--Mucoc  
--Atopy

The bo  
what ar

**Rosacea**

--Sebor  
--Atopy

*What are the classic ne*

**--Midface erythema**

--?

--?

**What is the classic trigger for worsening facial erythema in rosacea?**

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Consumption of

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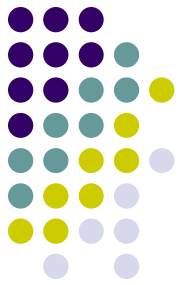
*What are the classic ne*  
**--Midface erythema**

--?  
--?

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Consumption of alcohol



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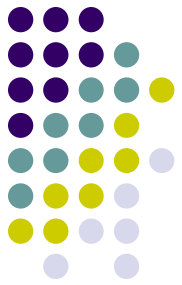
**--Midface erythema**

--?

--?

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Consumption of alcohol (**honorable mention if you said consumption of** two words )

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**Rosacea**

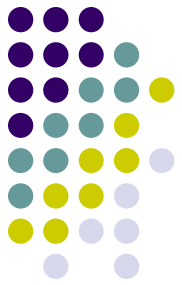
--Sebor  
--Atopy

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**--Midface erythema**

--?  
--?

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Consumption of alcohol (honorable mention if you said consumption of spicy food )

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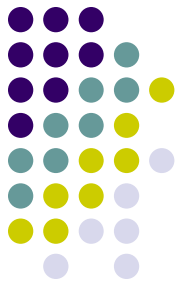
**Rosacea**

*What are the classic nonocular findings on exam?*  
--Midface erythema  
--?  
--?

← **Next finding**

Extrinsic  
orative  
Eye

# Dry Eye Syndrome



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The bo  
what ar

**Rosacea**

--Seb  
--Atopy

*What are the classic nonocular findings on exam?*

--Midface erythema

--Pustules/papules

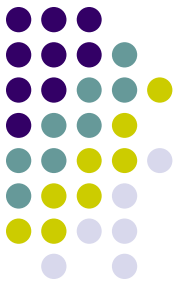
--?



Extrinsic

orative  
Eye

# Dry Eye Syndrome



Rosacea: Papules/pustules

# Dry Eye Syndrome



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*What are the classic nonocular findings on exam?*

--Midface erythema

--Pustules/papules

--Thickening of nasal skin (called )

Extrinsic

orative  
Eye

The Co  
what ar  
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*Meibomian gland* Widened

Reduced blink rate

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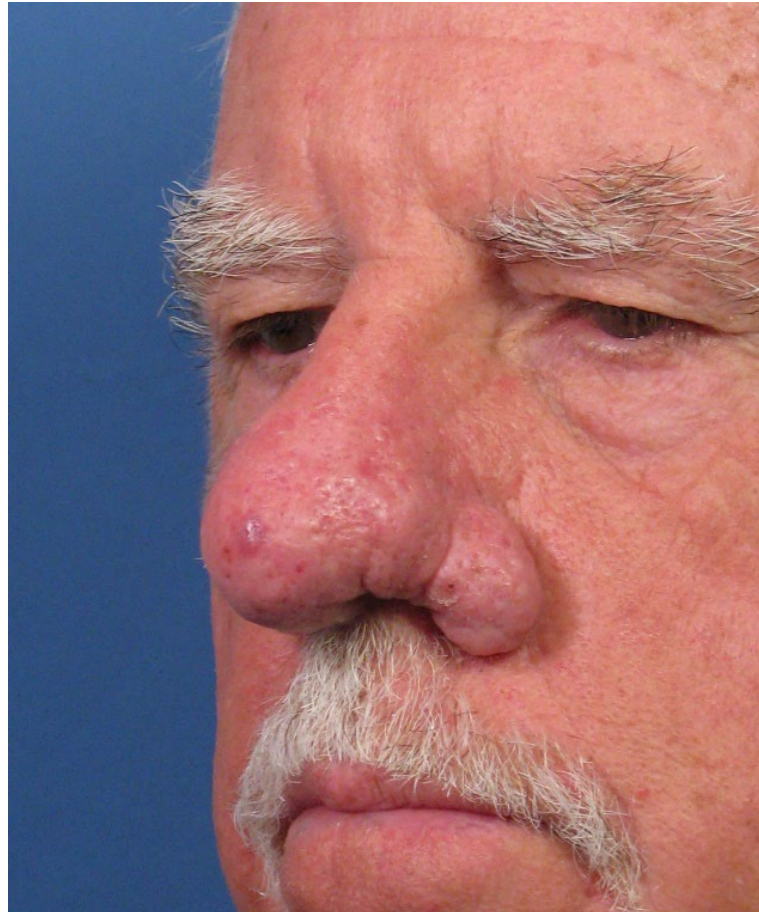
The bo  
what ar  
--Rosacea  
--Sebor  
--Atopy

**Rosacea**

*What are the classic nonocular findings on exam?*  
--Midface erythema  
--Pustules/papules  
--Thickening of nasal skin (called *rhinophyma*)

Extrinsic  
orative  
Eye

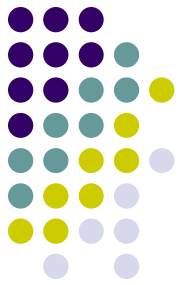
# Dry Eye Syndrome



Rosacea: Rhinophyma



# Dry Eye Syndrome



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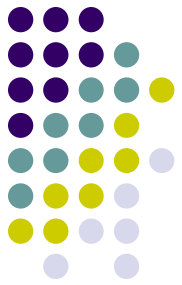
--Midface erythema

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*What does the Cornea book call “the mainstay of therapy” for rosacea?*

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Oral



Extrinsic

orative  
Eye

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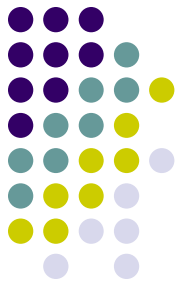
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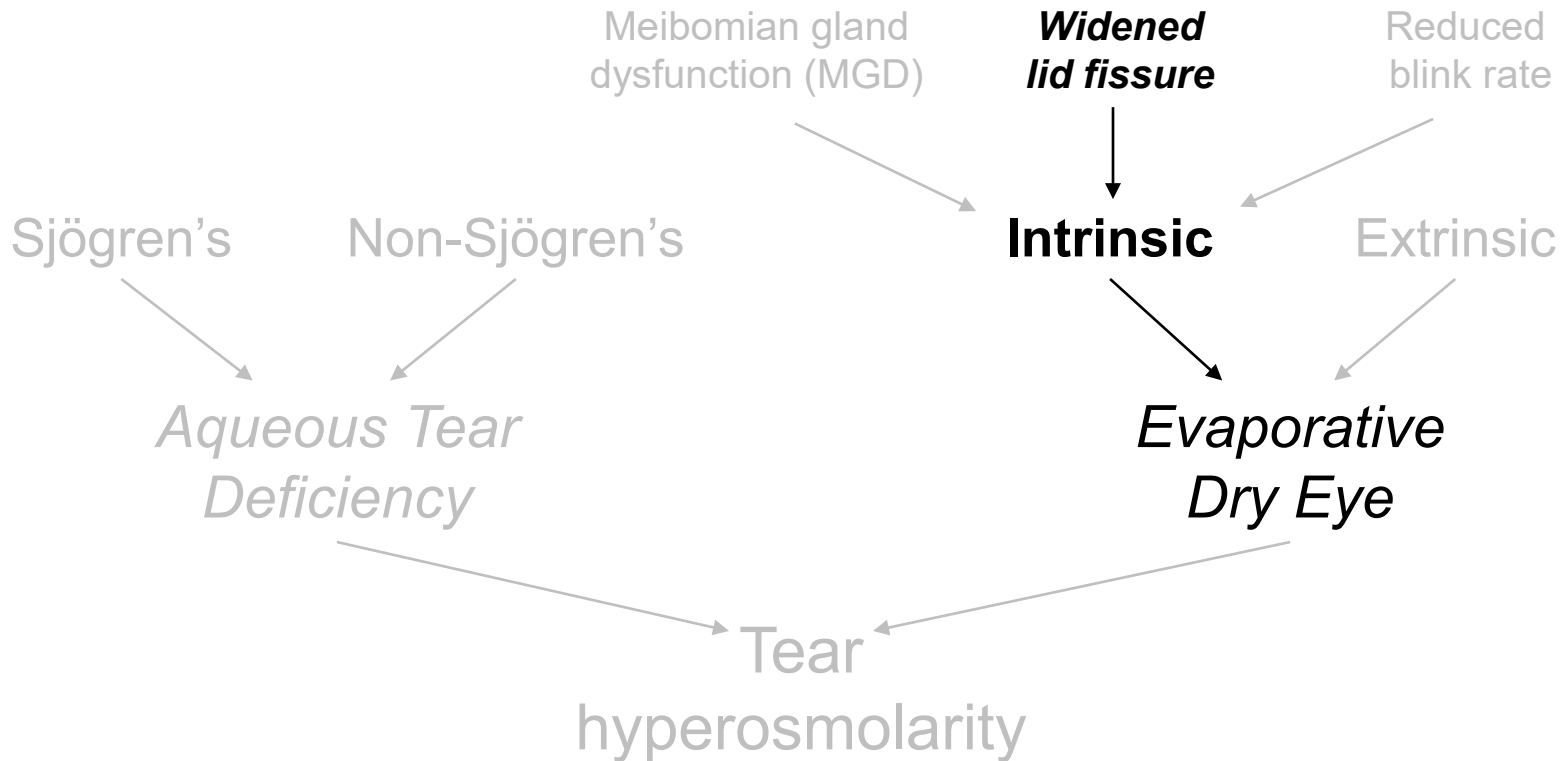
Oral tetracyclines

# Dry Eye Syndrome



What are the causes of a widened lid fissure?

- ?
- ?
- ?



# Dry Eye Syndrome

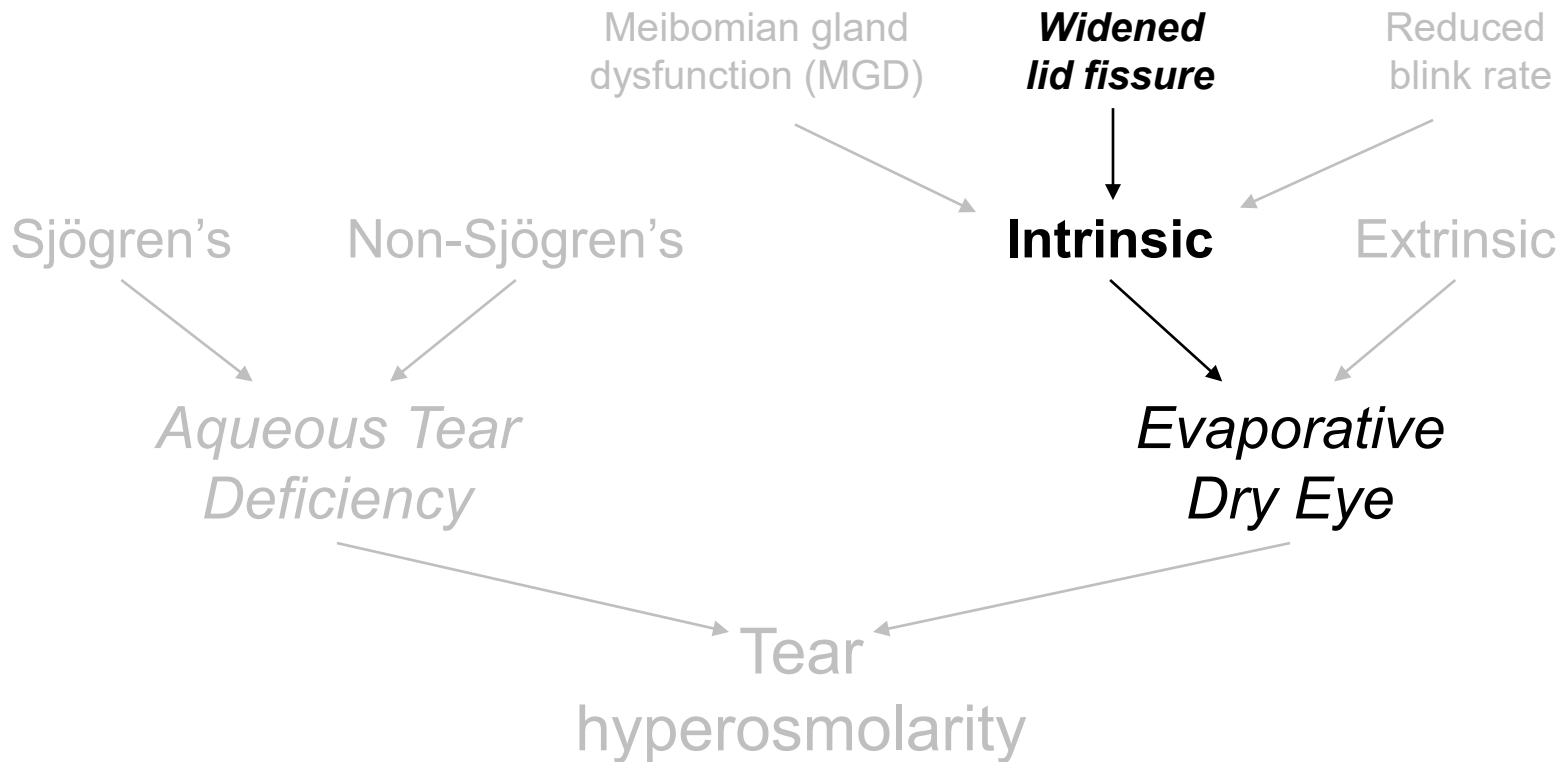


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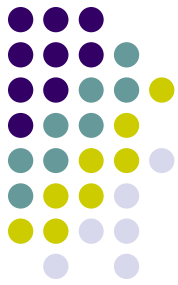
--Forward displacement of the globe (ie, proptosis/exophthalmos)

--?

--?



# Dry Eye Syndrome



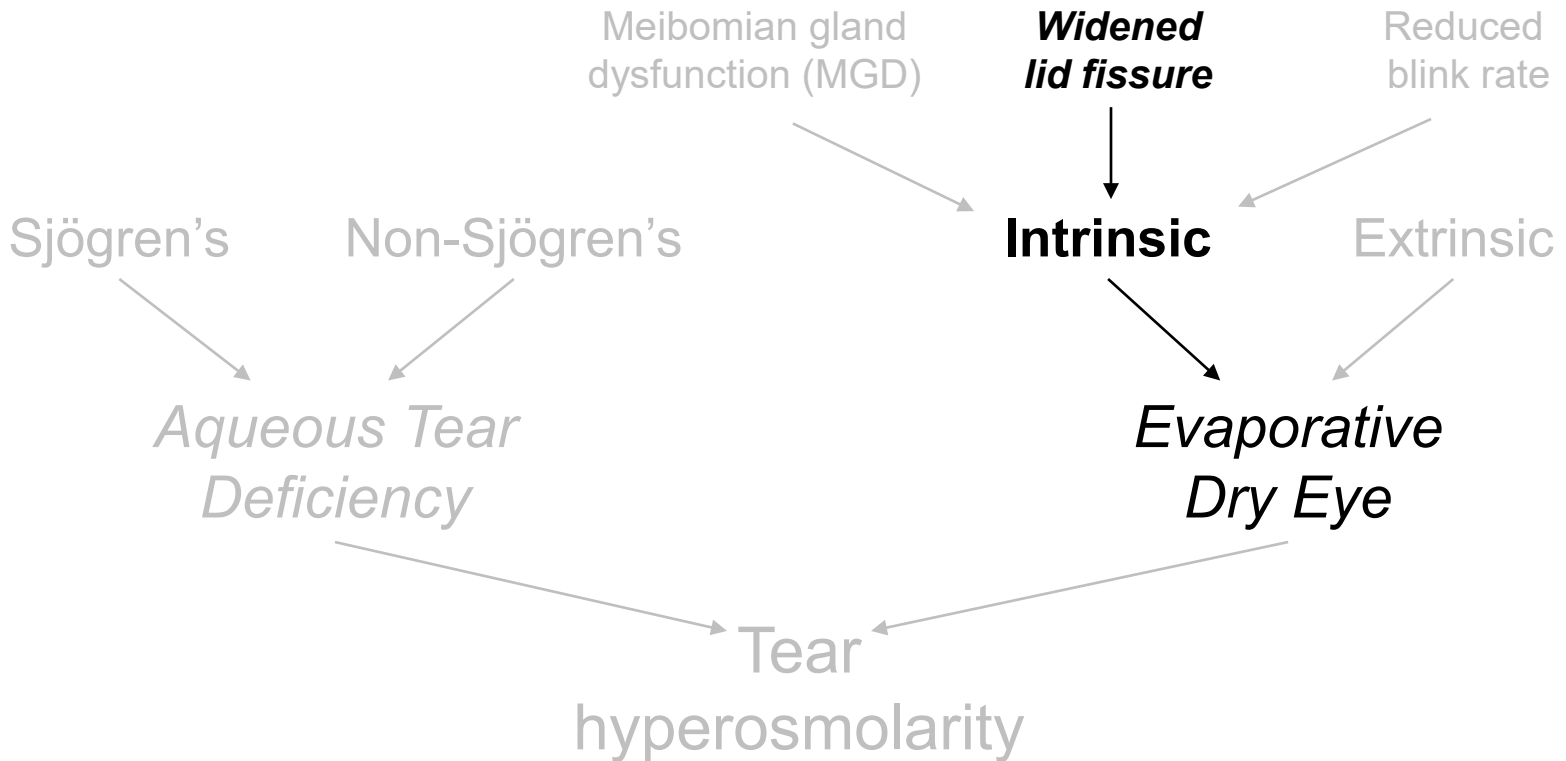
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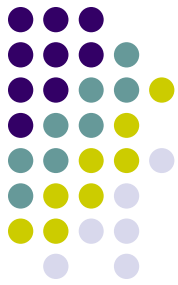
--Increased innervation to the lid retractors such as occurs in

three words

--?

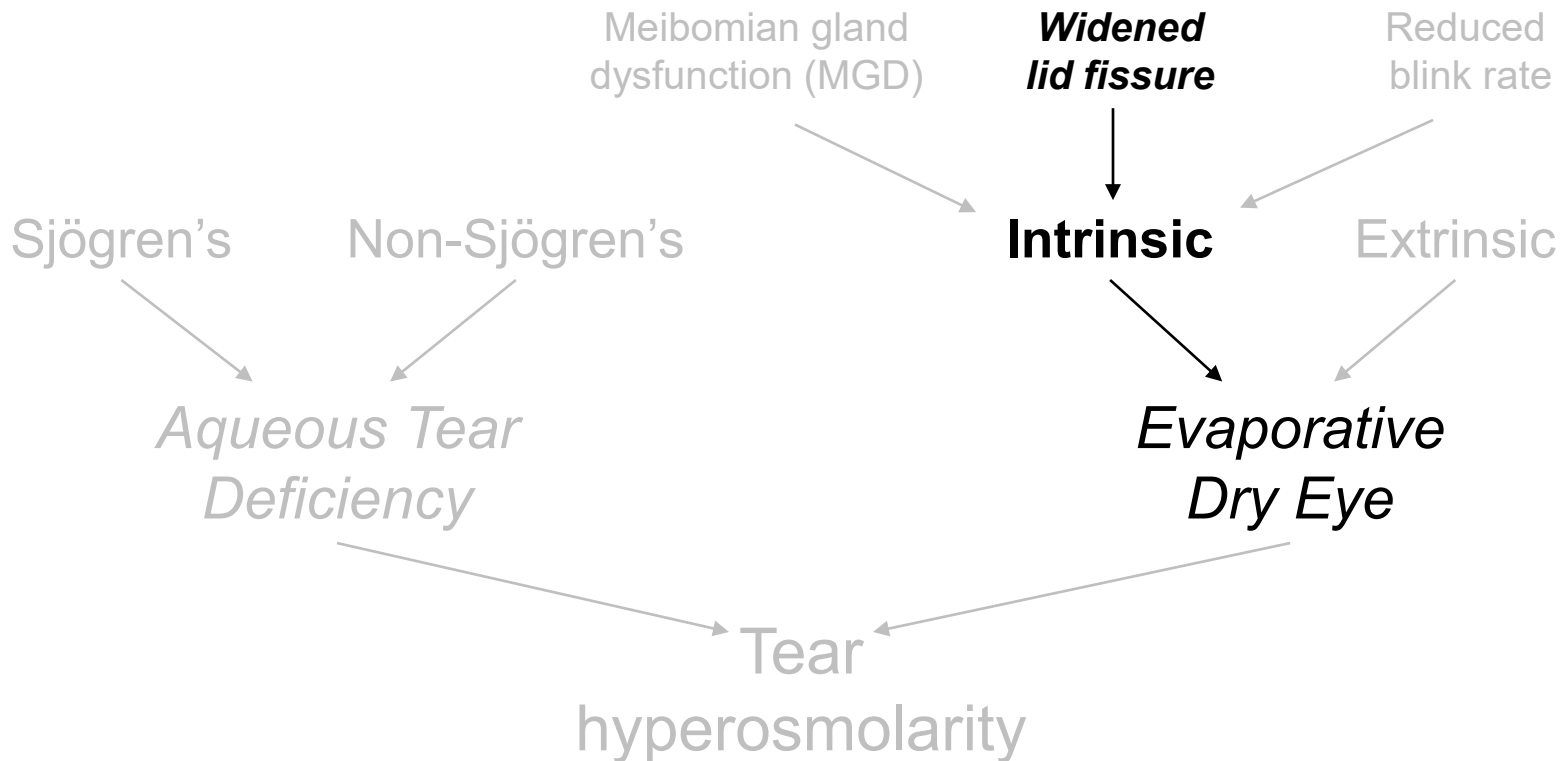


# Dry Eye Syndrome



*What are the causes of a widened lid fissure?*

- Forward displacement of the globe (ie, proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- ?

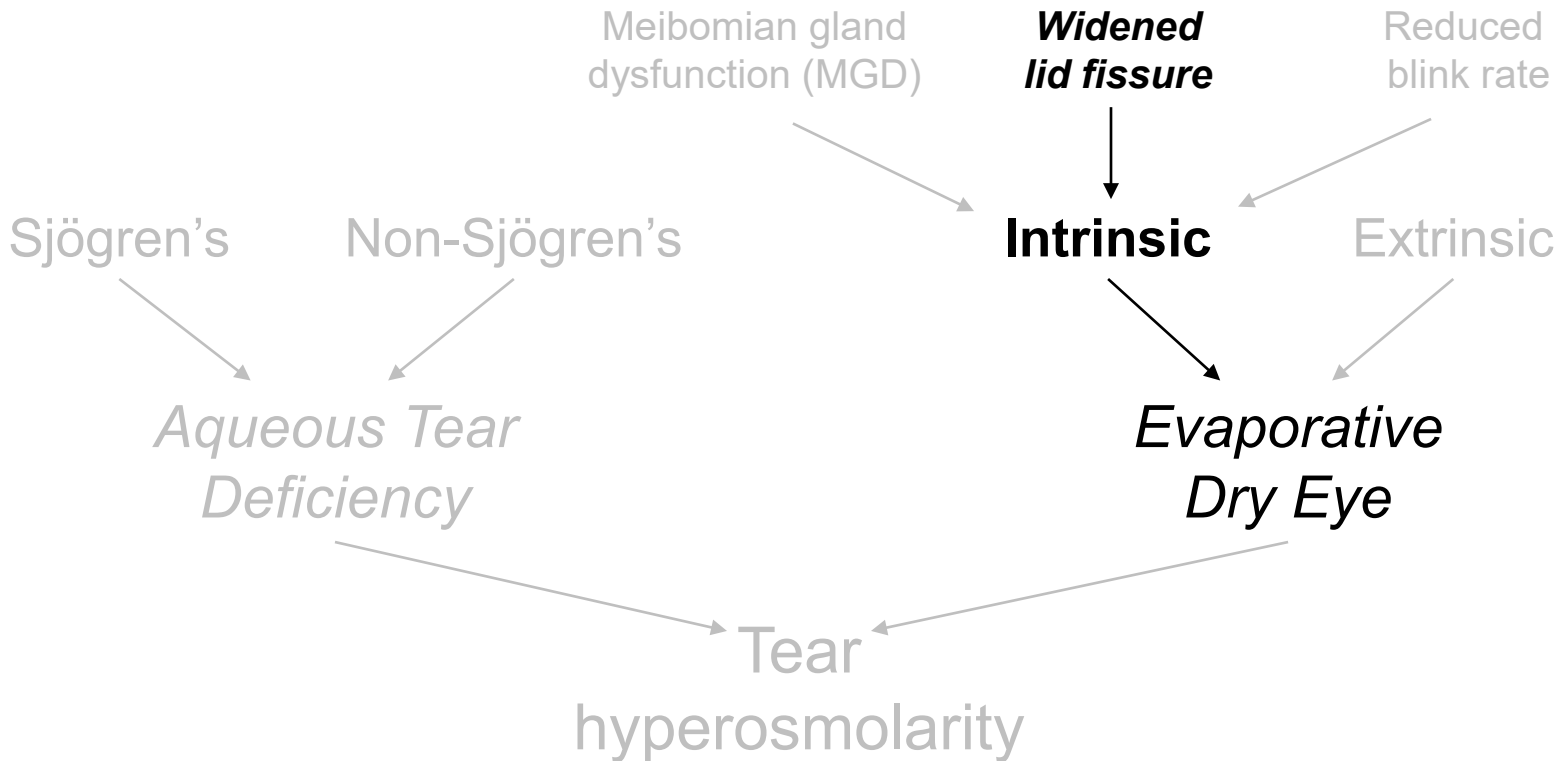


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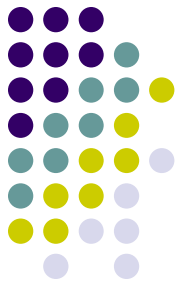
*What are the causes of a widened lid fissure?*

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- Congenital craniofacial malformations resulting in  orbits



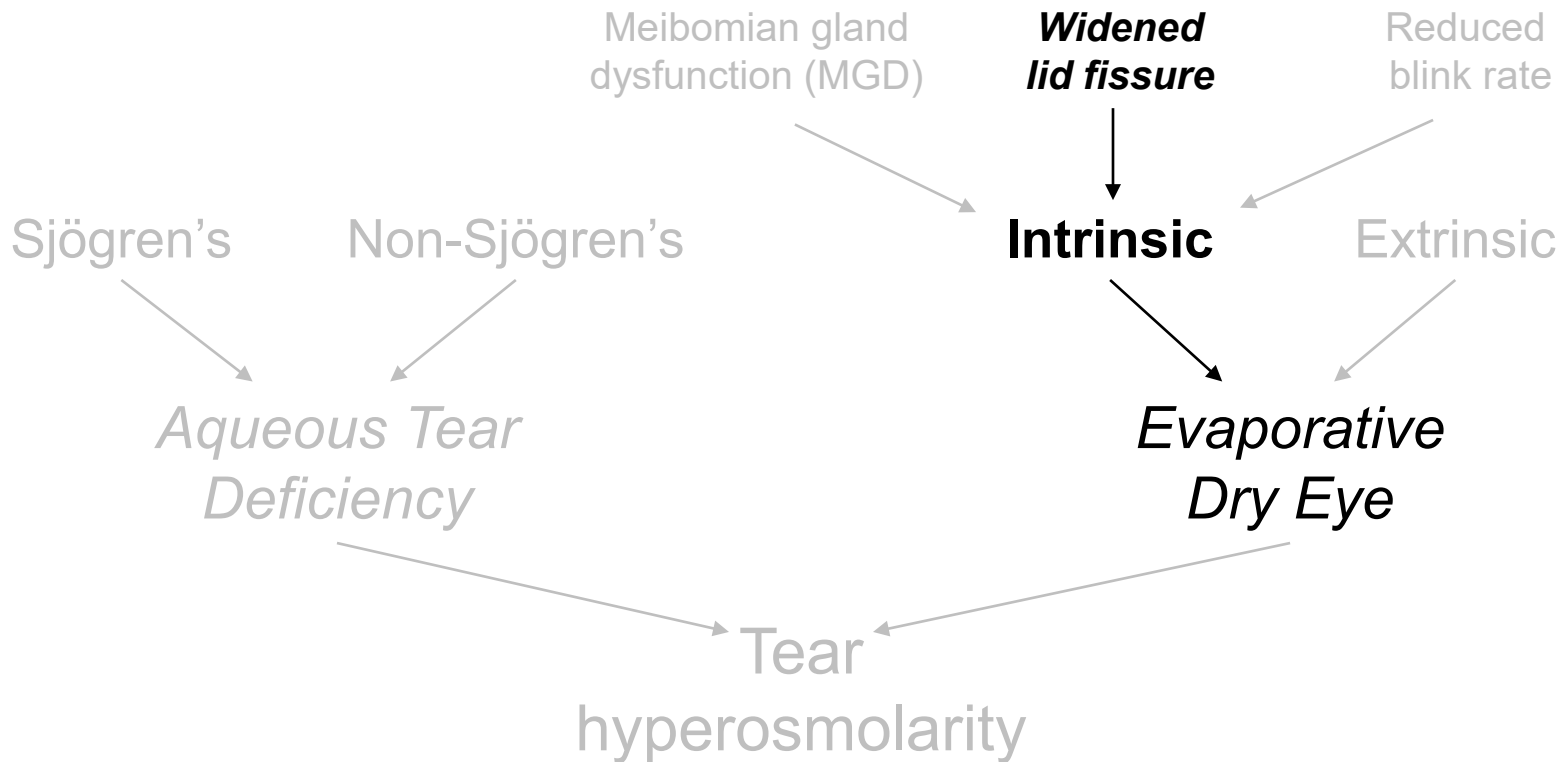


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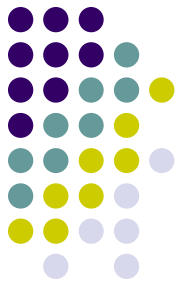


*What are the causes of a widened lid fissure?*

- Forward displacement of the globe (ie, proptosis/exophthalmos)
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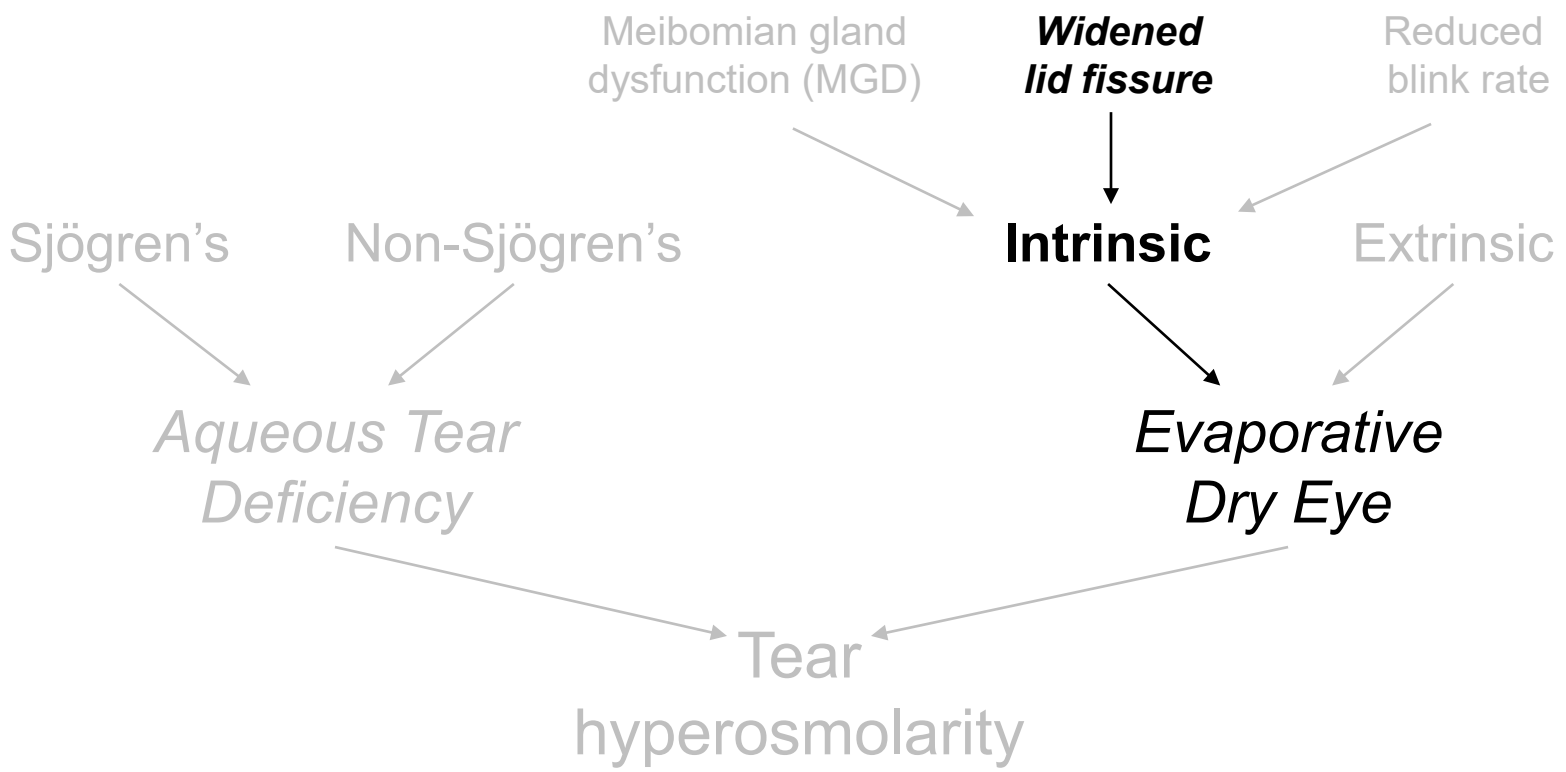


# Dry Eye Syndrome



What group of congenital craniofacial malformations are strongly associated with shallow orbits?

- widened lid fissure?
- proptosis (ie, globe protrusion)
- increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits**

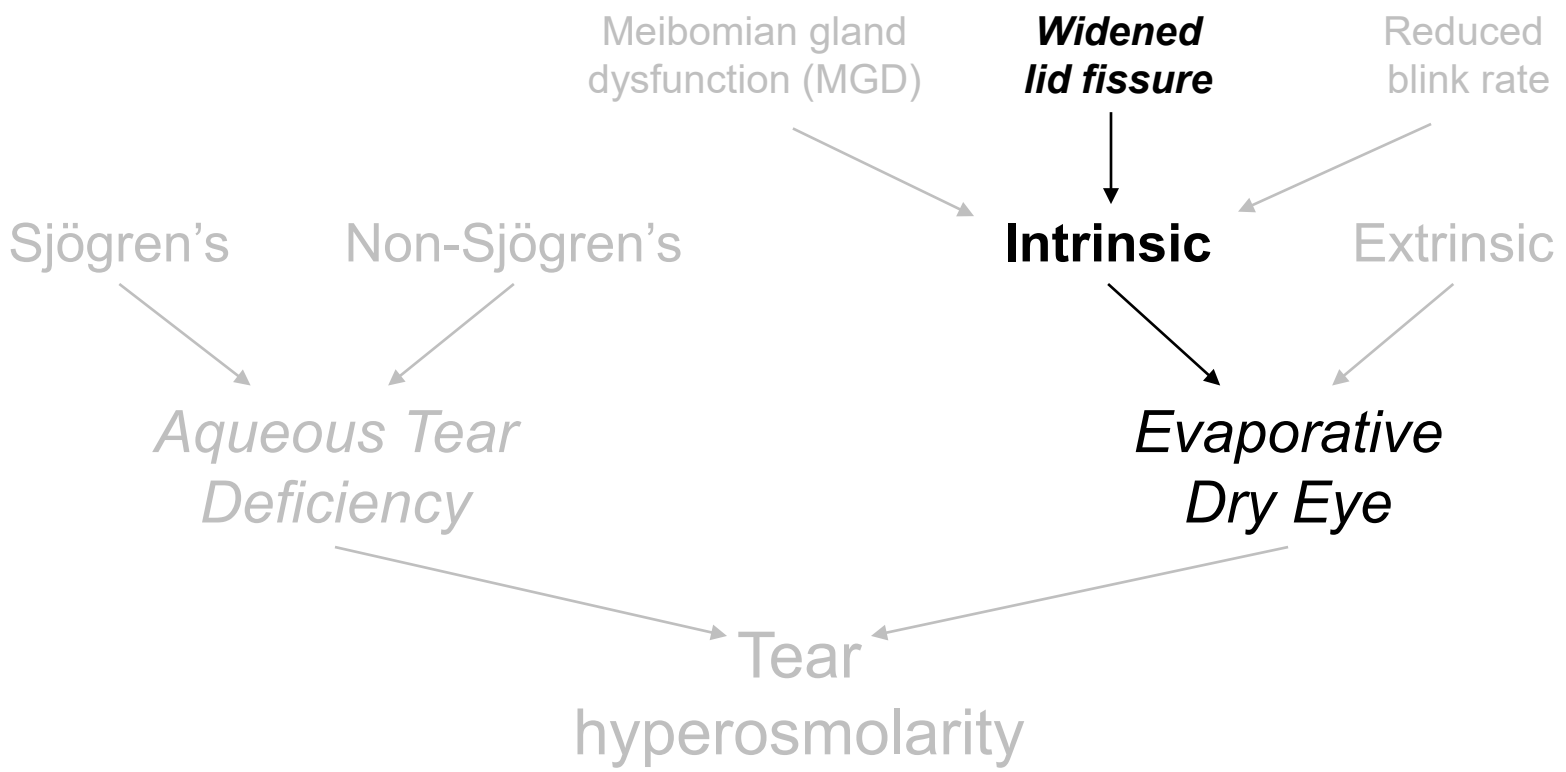


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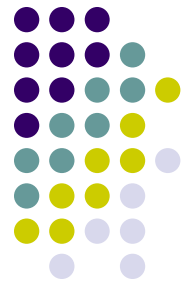


What group of congenital craniofacial malformations are strongly associated with shallow orbits?  
The craniosynostoses

- widened lid fissure?
- the globe (ie, proptosis/exophthalmos)
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# Dry Eye Syndrome



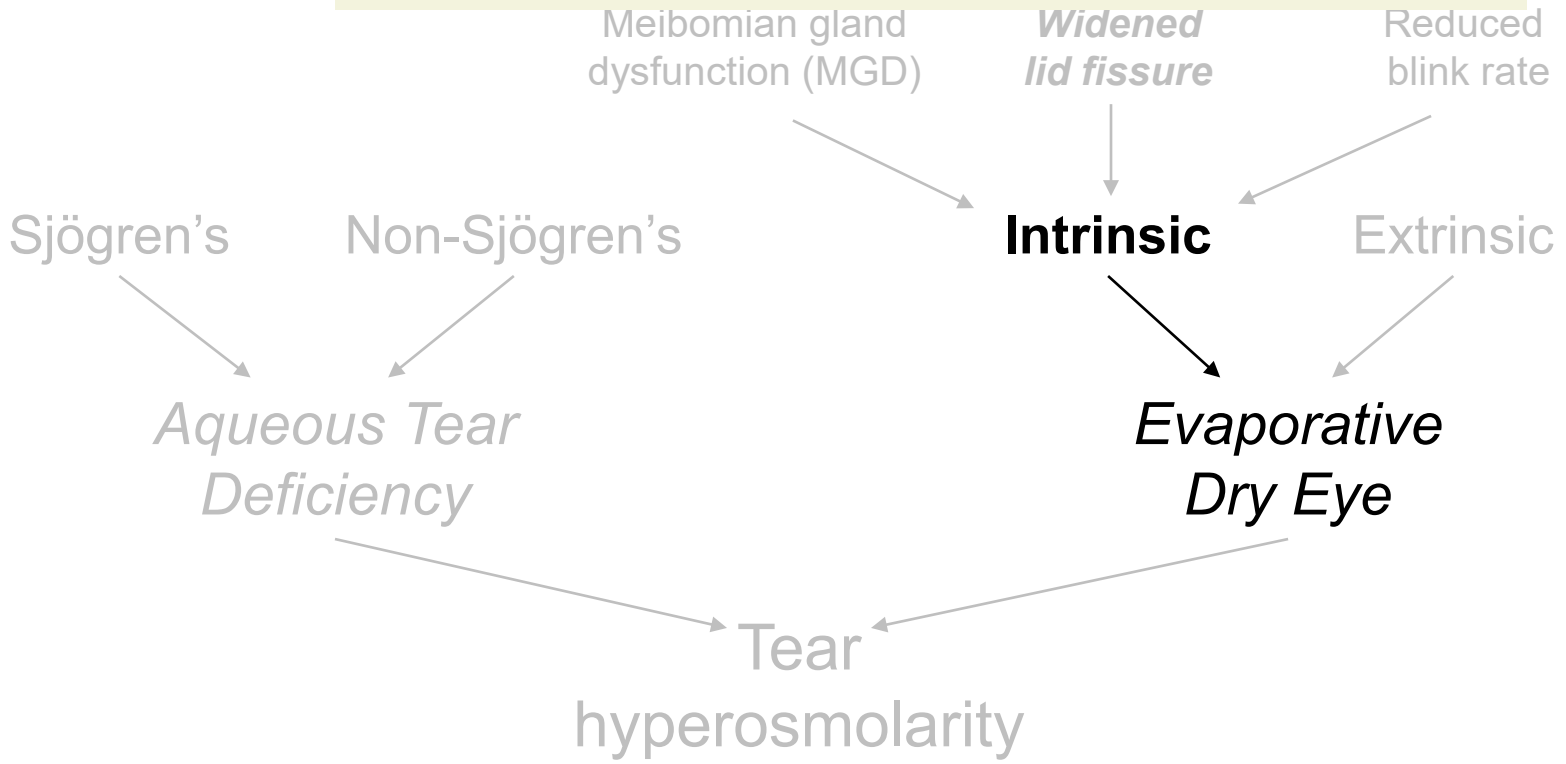
What group of congenital craniofacial malformations are strongly associated with shallow orbits?

The **craniosynostoses**

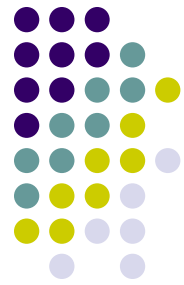
Widened lid fissure?

In a nutshell, what is the causal mechanism of the craniosynostoses?

the disease  
is



# Dry Eye Syndrome

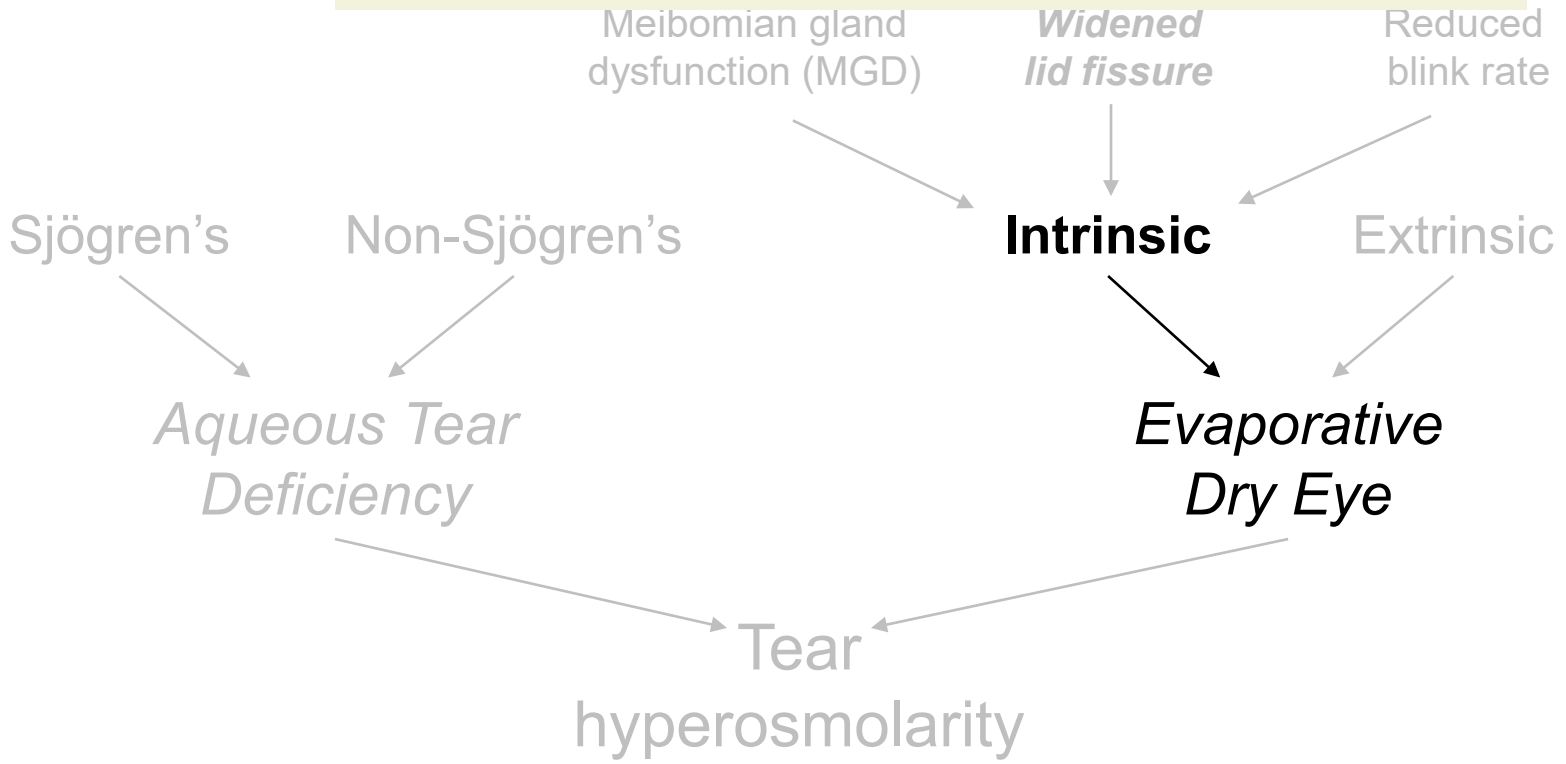


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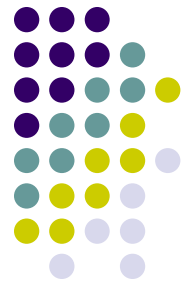
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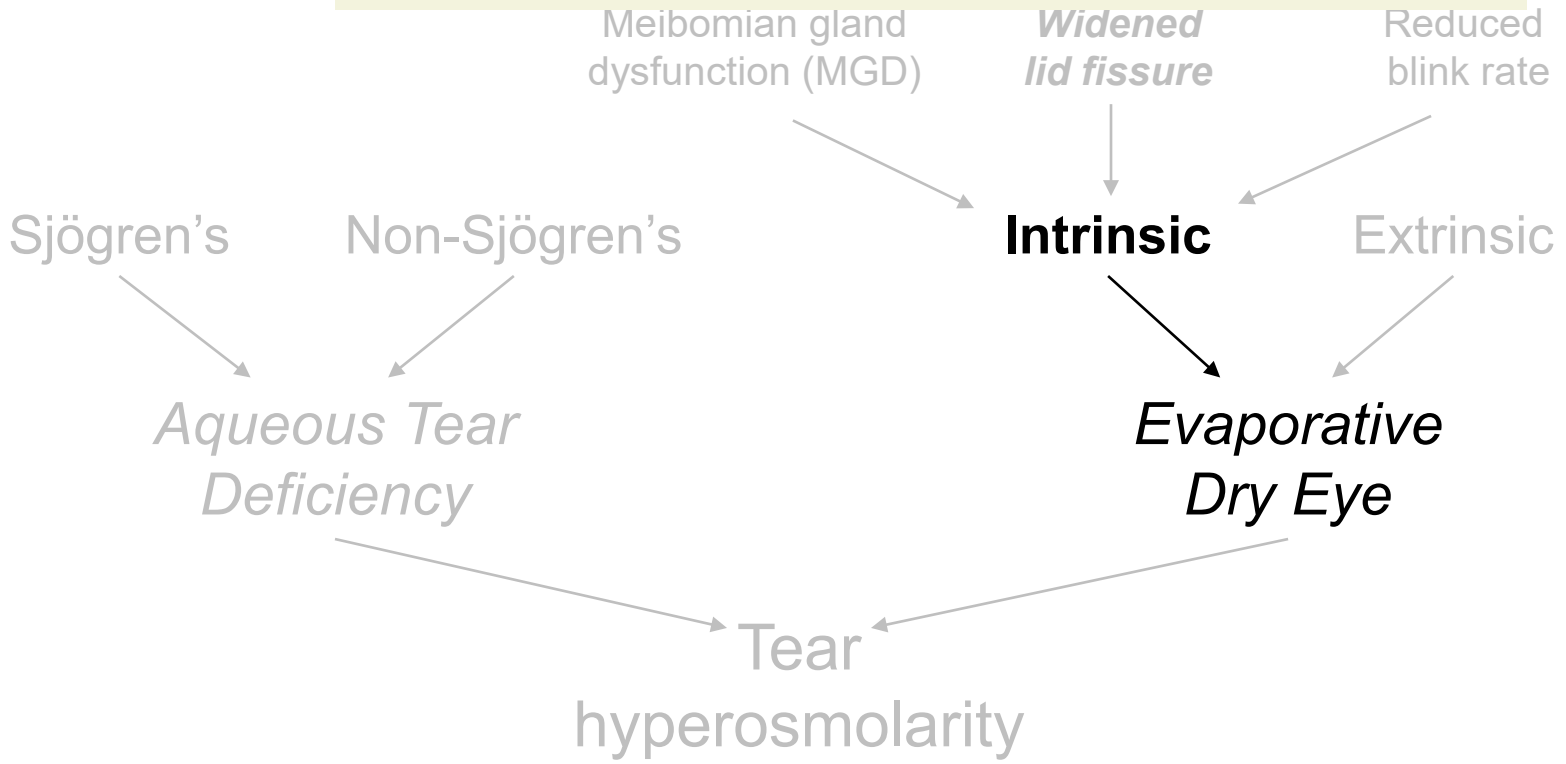


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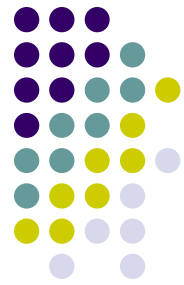
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...e disease  
...s



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What are the four craniosynostoses discussed in detail in the BCSC?

the disease  
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- ?
- ?
- ?
- ?

Meibomian gland dysfunction (MGD)

**Widened lid fissure**

Reduced blink rate

Sjögren's

Non-Sjögren's

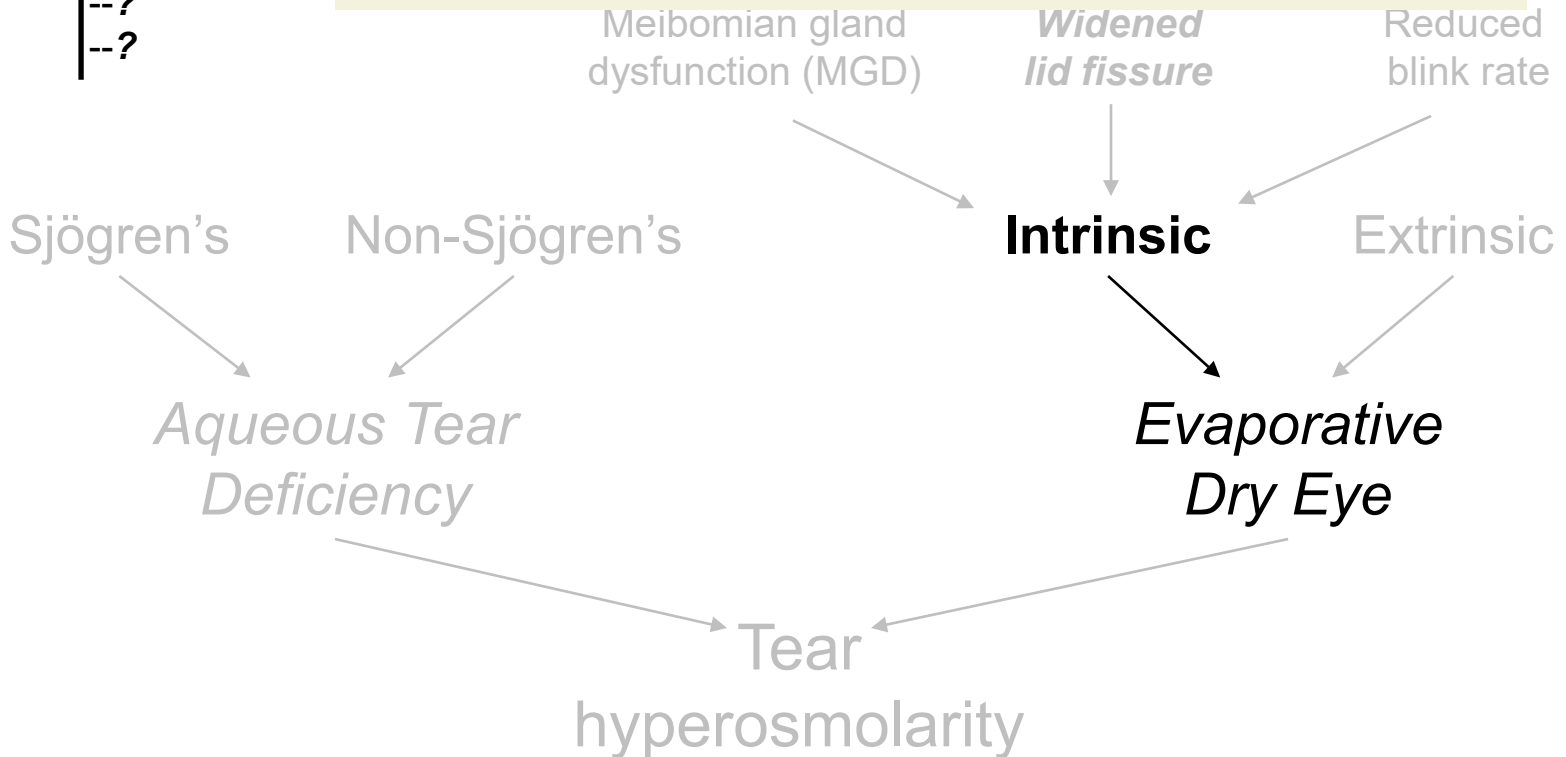
**Intrinsic**

Extrinsic

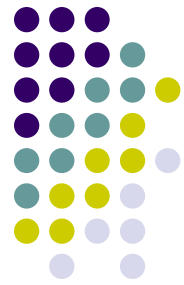
*Aqueous Tear Deficiency*

*Evaporative Dry Eye*

Tear hyperosmolarity



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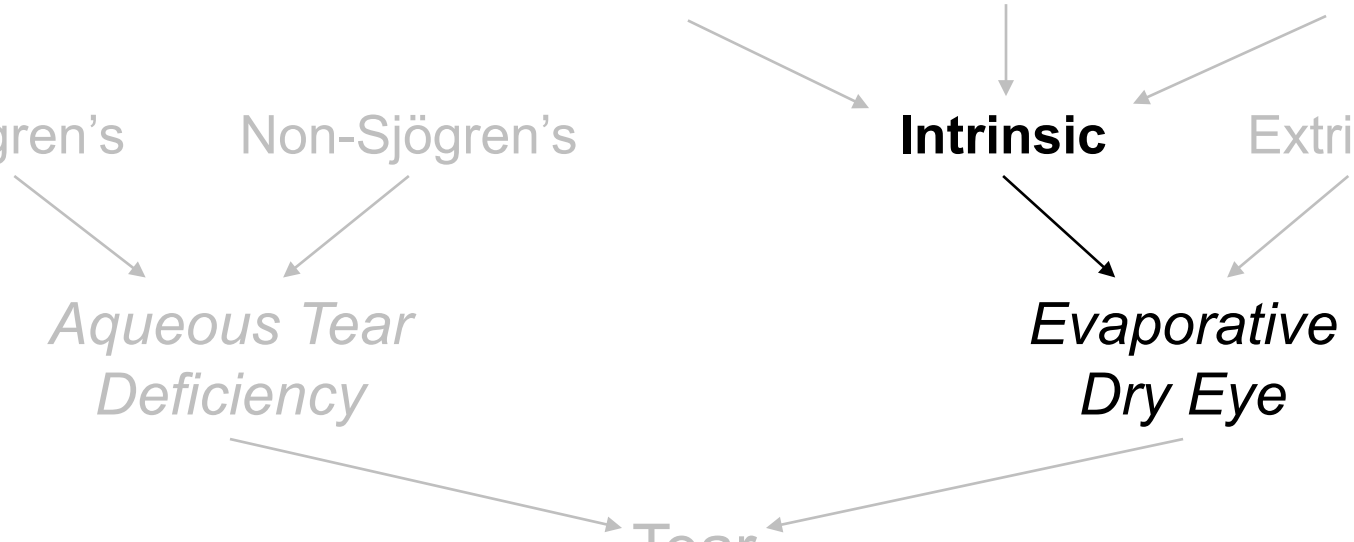
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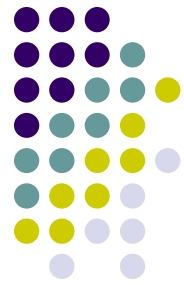
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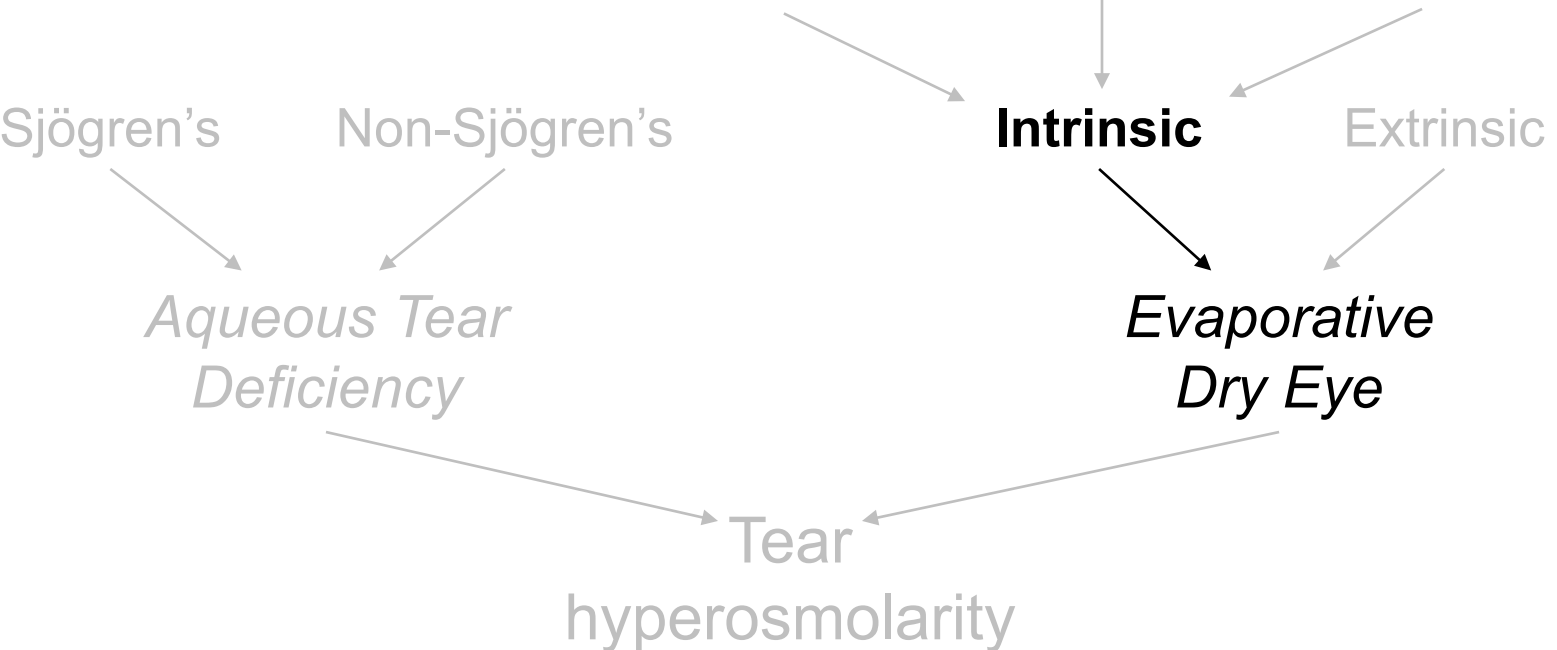
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BCSC?

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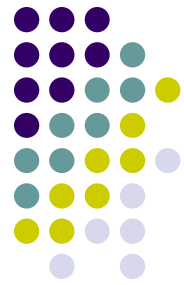
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Reduced blink rate

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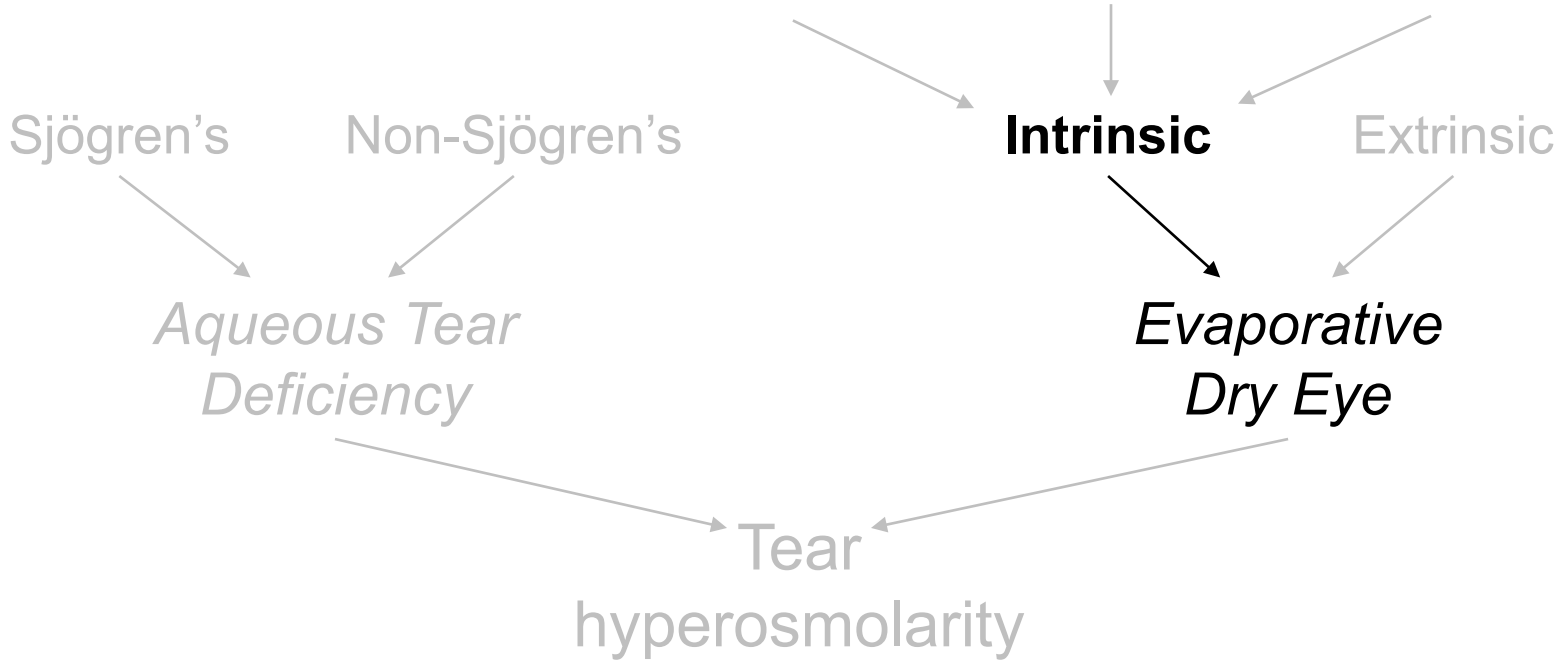
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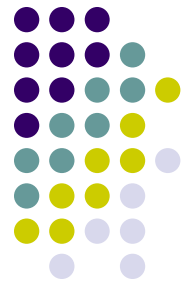
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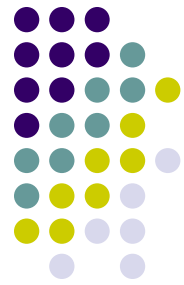
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**For more on the craniosynostoses, see slide-set P22**

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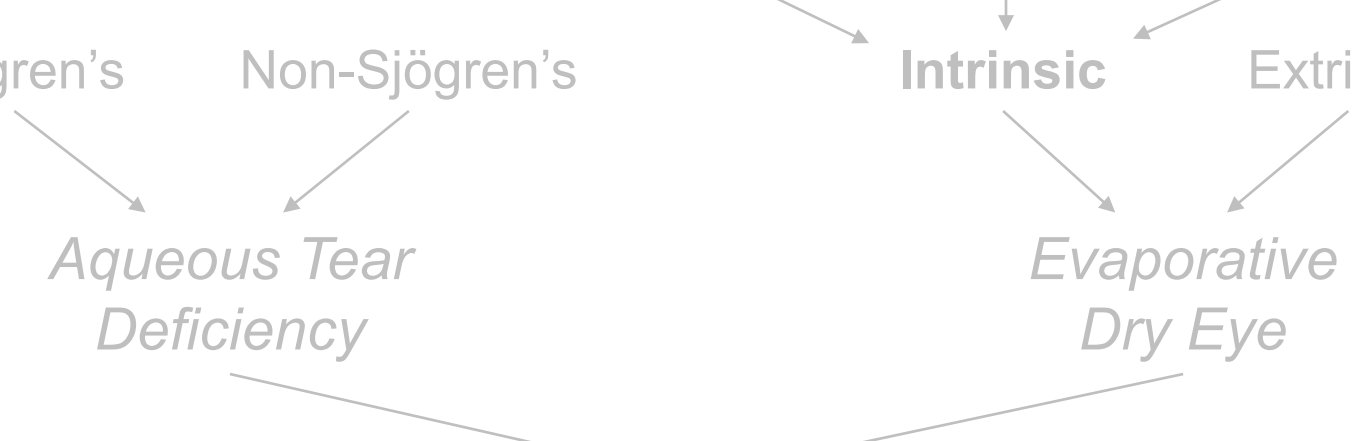
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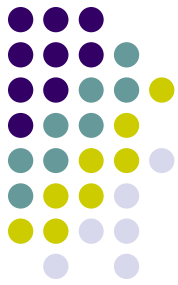
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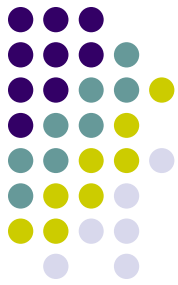
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Physiological (ie, a normal phenomenon), and pathological



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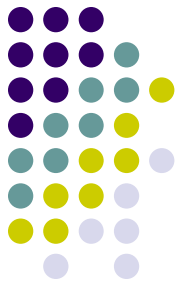
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What is the most common physiological cause of reduced blink rate?





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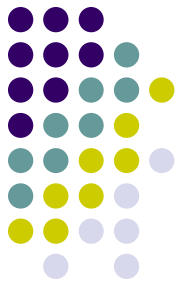


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# Dry Eye Syndrome



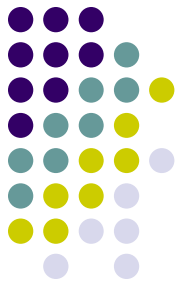
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What is the most common physiological cause of reduced blink rate?  
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What is the most common pathological cause of reduced blink rate?  
Parkinson's dz



# Dry Eye Syndrome



The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**

Recall that earlier in the set we alluded to a *third* means by which tear-film status could produce hyperosmolarity and dry eye. The time to address this has arrived!

**Head's up:** Later in the set we're gonna add a *third* mechanism leading to tear hyperosmolarity

*three*  
In what ~~two~~ fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

*Aqueous Tear  
Deficiency*

*Evaporative  
Dry Eye*

→ Tear  
hyperosmolarity ←

## Dry Eye Syndrome



The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

*In what **other** fundamental way could the status of the tear film lead to tear hyperosmolarity?*

--?

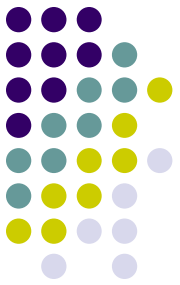
*Aqueous Tear  
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Dry Eye*

Tear  
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A diagram showing two arrows pointing towards the text 'Tear hyperosmolarity'. One arrow originates from the text 'Aqueous Tear Deficiency' and the other from 'Evaporative Dry Eye'.

## Dry Eye Syndrome



The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

*In what **other** fundamental way could the status of the tear film lead to tear hyperosmolarity?*

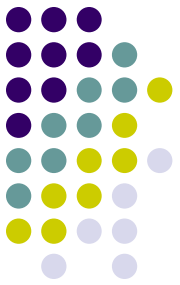
--The tear film can two words too quickly, exposing the ocular surface.

*Aqueous Tear  
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Tear  
hyperosmolarity

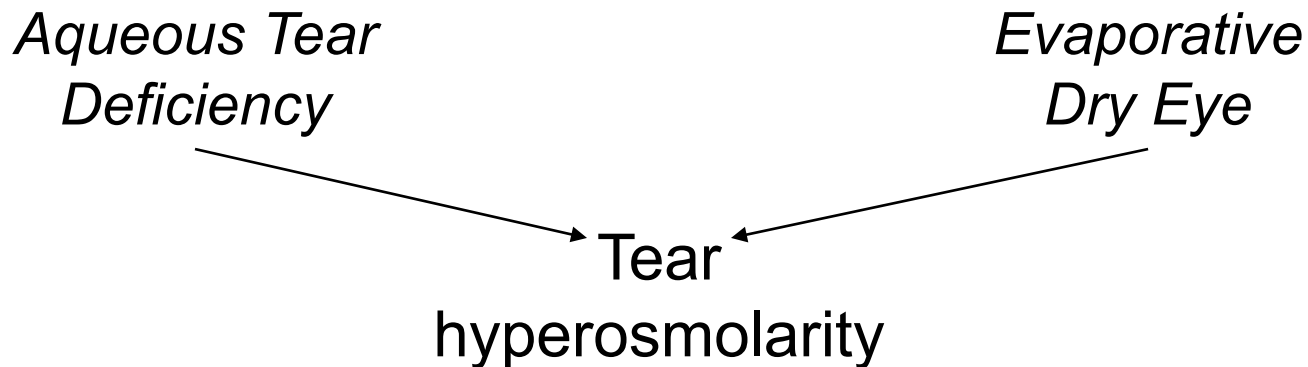
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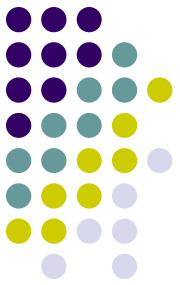
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*In what **other** fundamental way could the status of the tear film lead to tear hyperosmolarity?*

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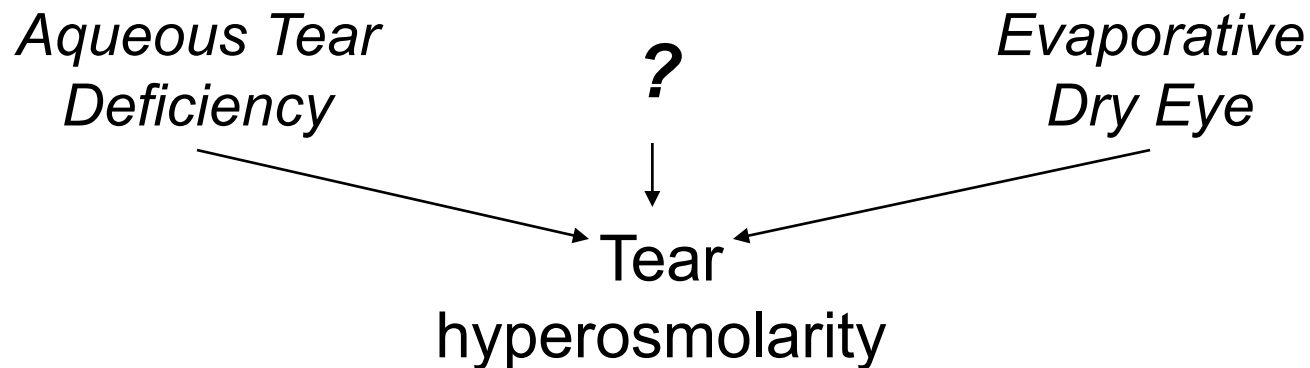
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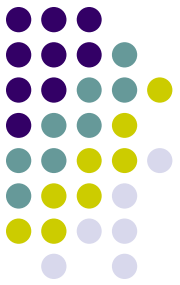
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**This state is known as one of...**





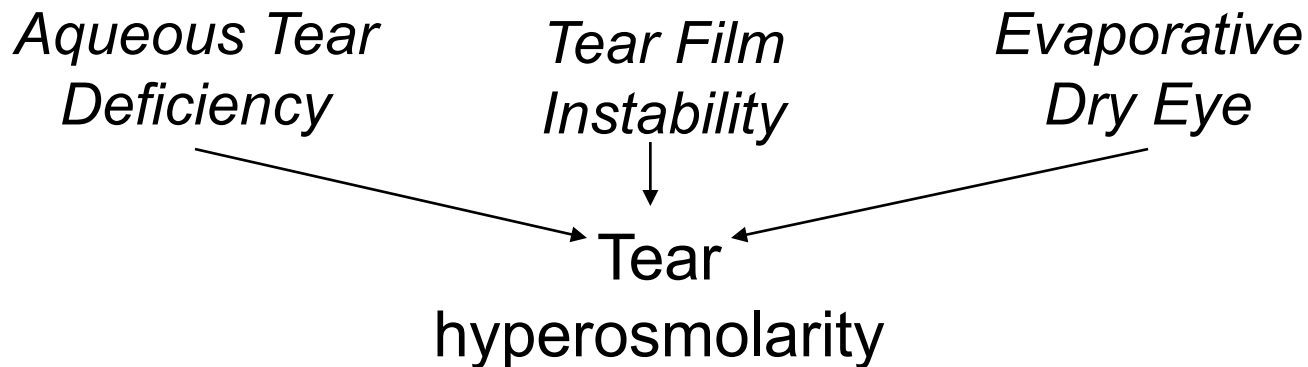
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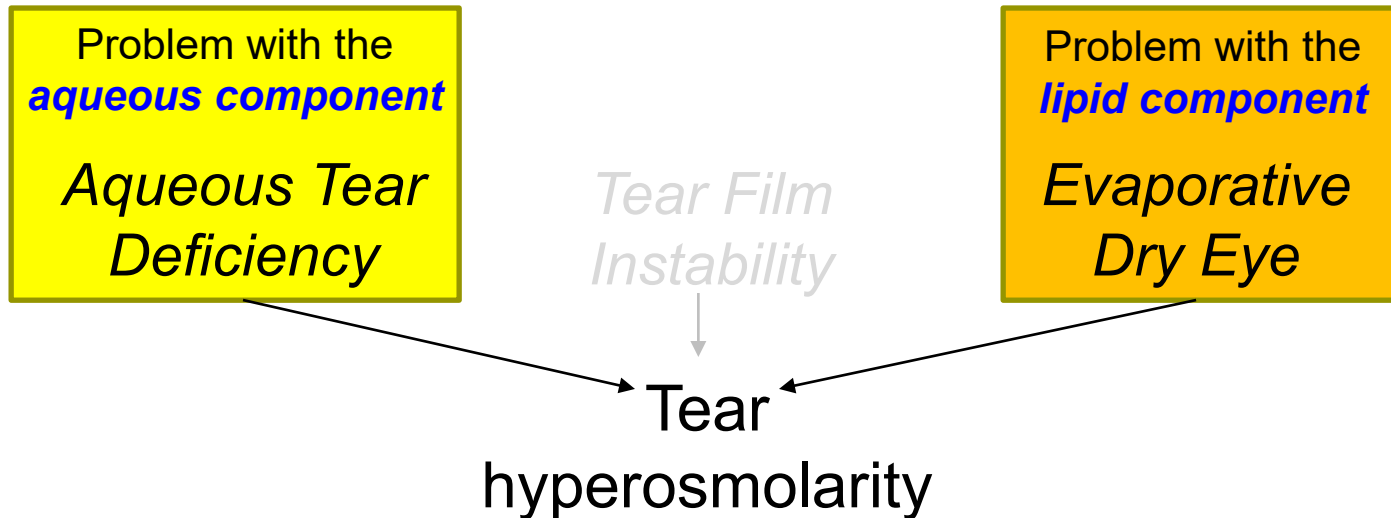


The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

Recalling our answers to **this** issue previously:



*While it's a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:*



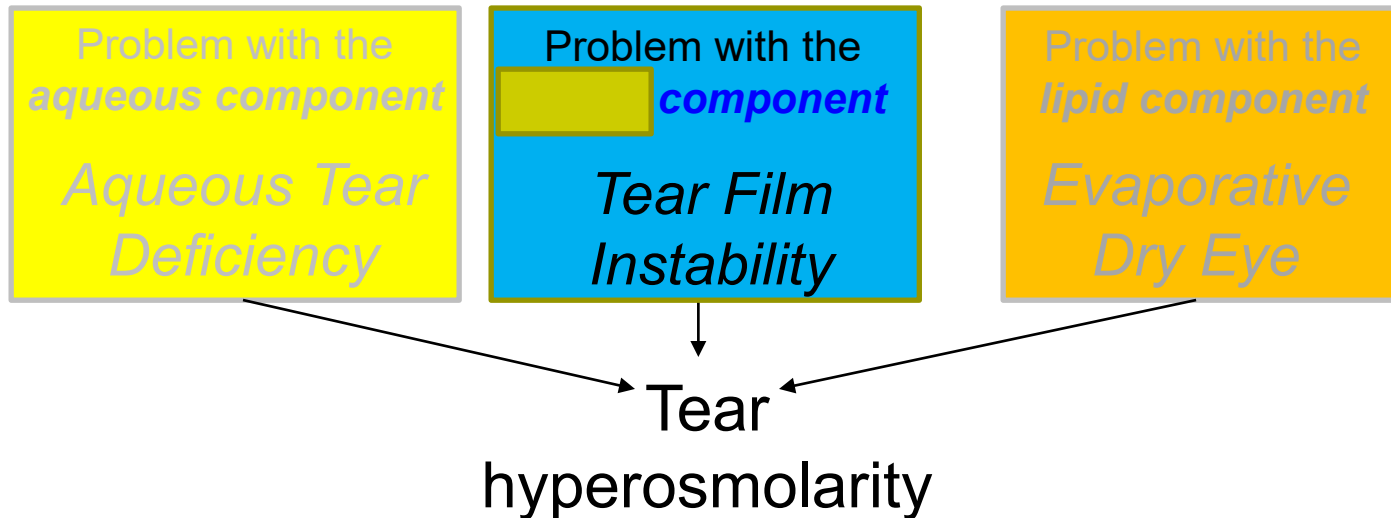
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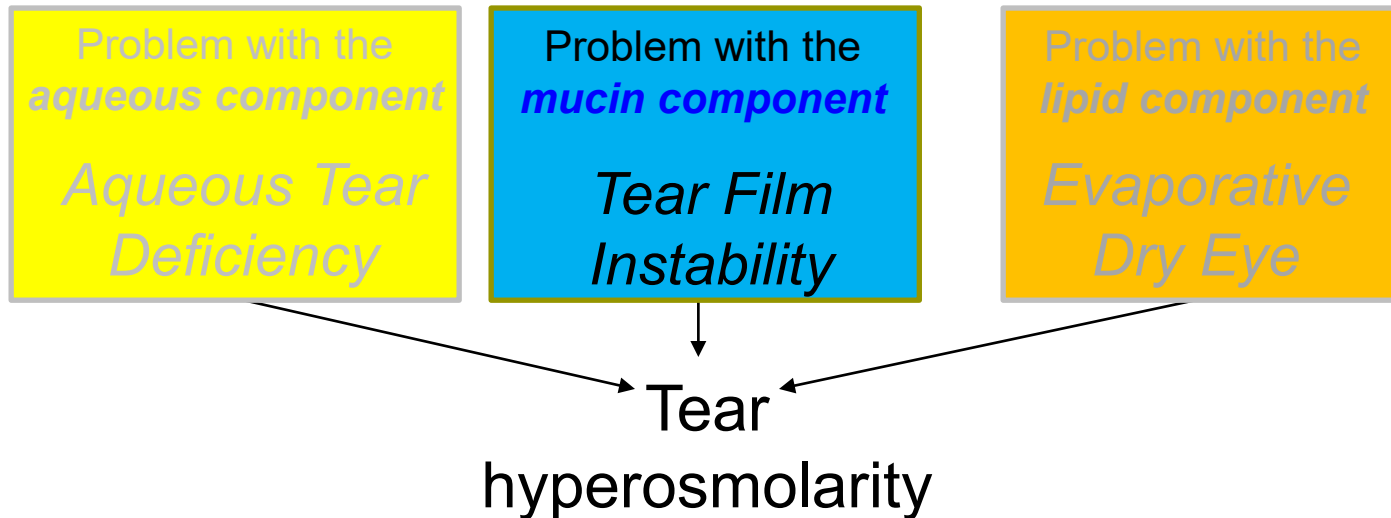
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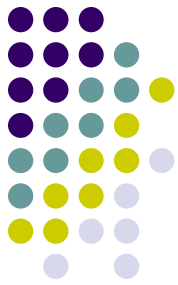
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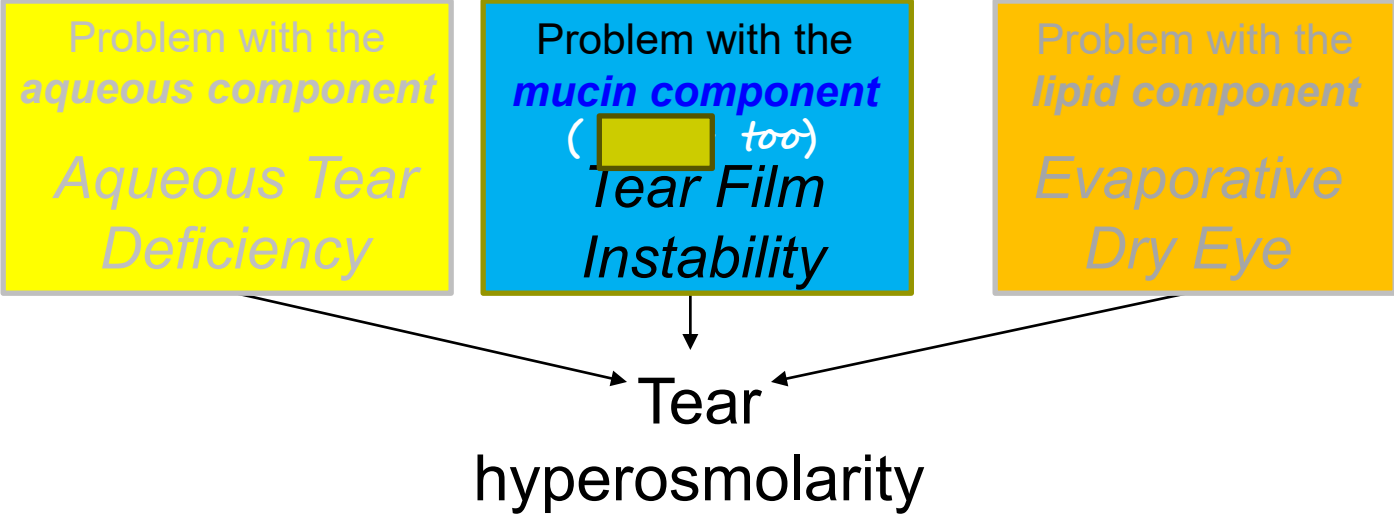
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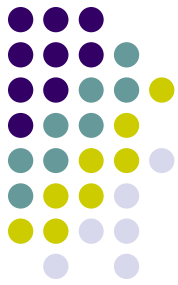
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One important oversimplification to note is the implication below that tear-film instability is a function **only** of the mucin component, when in fact the status of the **lipid** component makes a significant contribution to tear-film (in)stability as well

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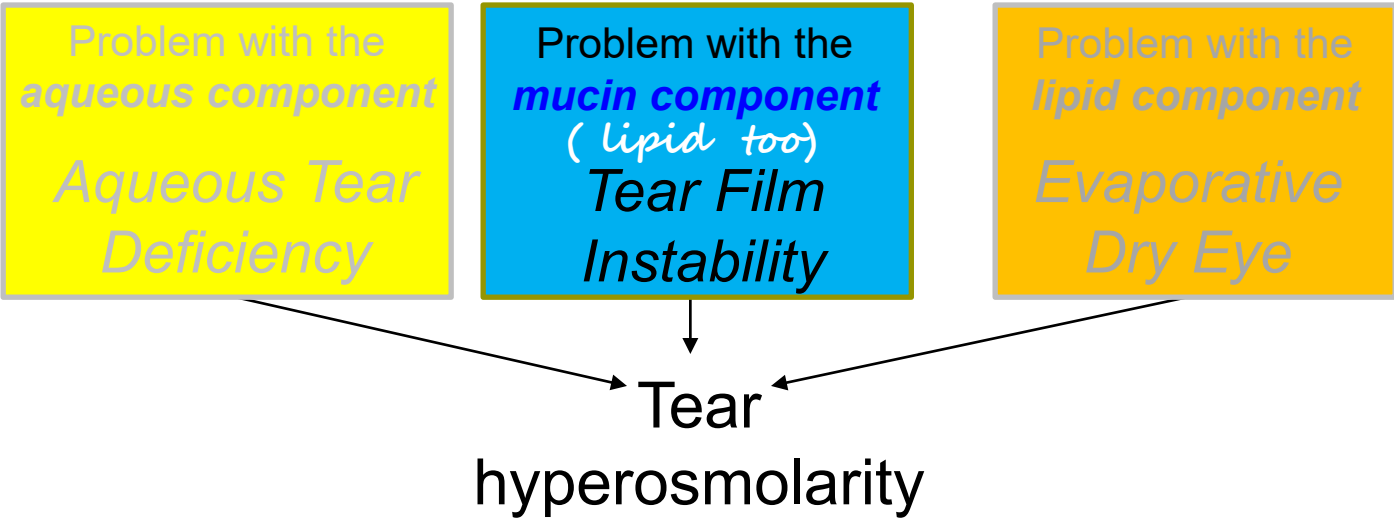
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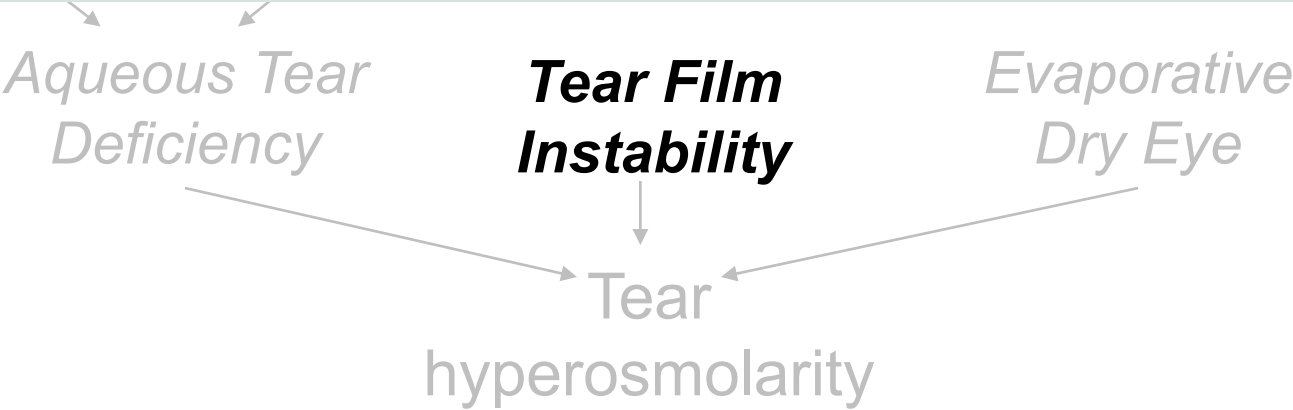
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# Dry Eye Syndrome



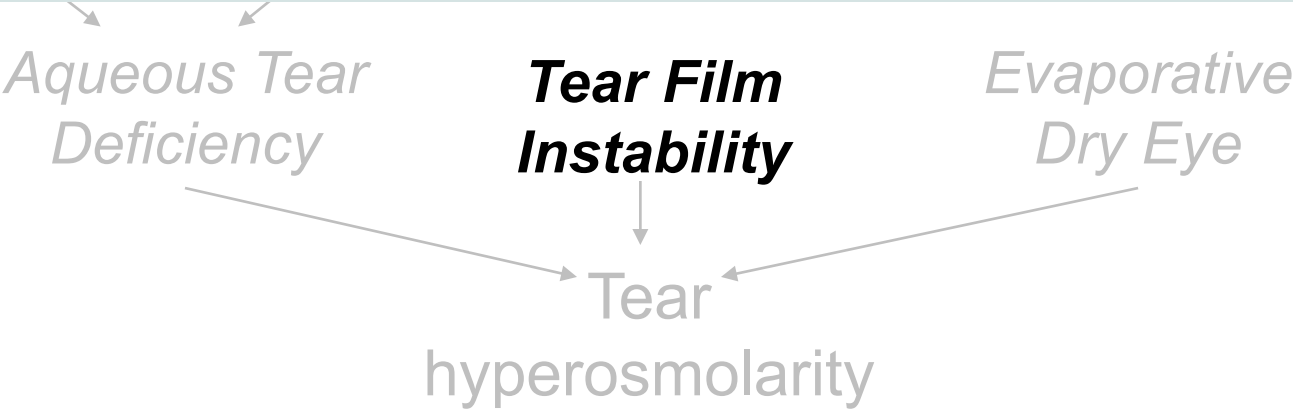
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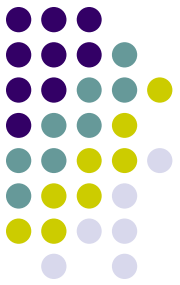


How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?  
The **tear-film break-up time (TBUT or TFBUT)** assessment





# Dry Eye Syndrome



*How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?*

The **tear-film break-up time (TBUT or TFBUT)** assessment

*How is TBUT assessed, ie, what are the steps involved?*

*Aqueous Tear  
Deficiency*

***Tear Film  
Instability***

*Evaporative  
Dry Eye*

Tear  
hyposmolarity



# Dry Eye Syndrome

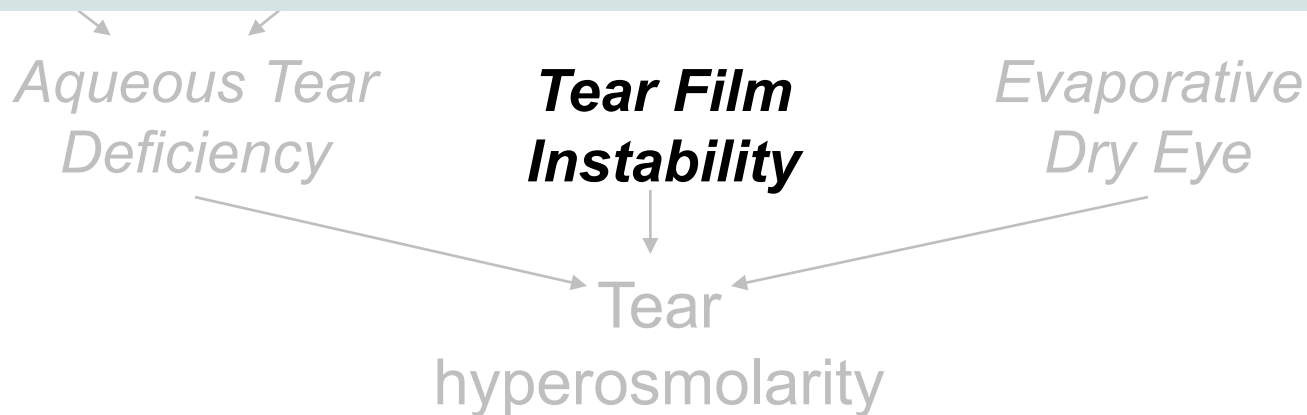


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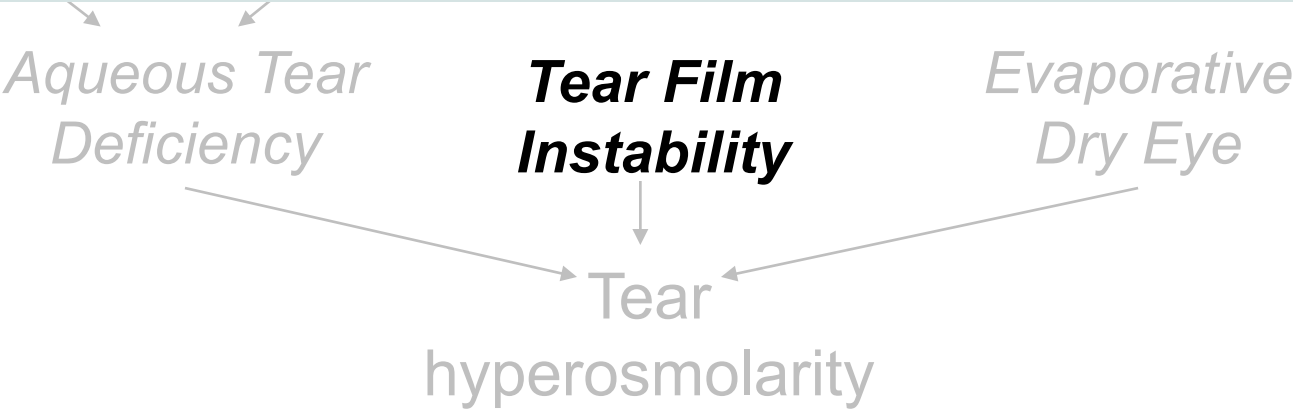


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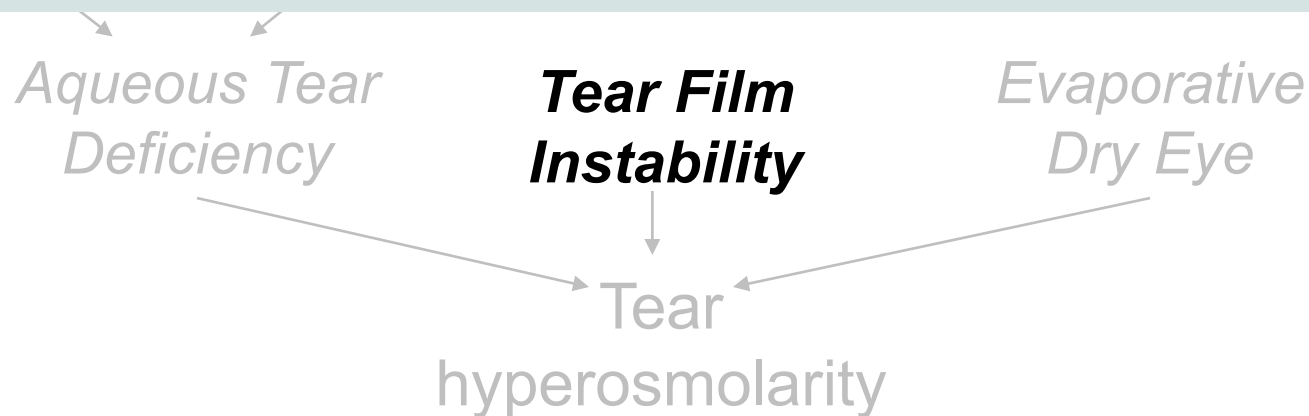


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The **tear-film break-up time** (*TBUT* or *TFBUT*) assessment

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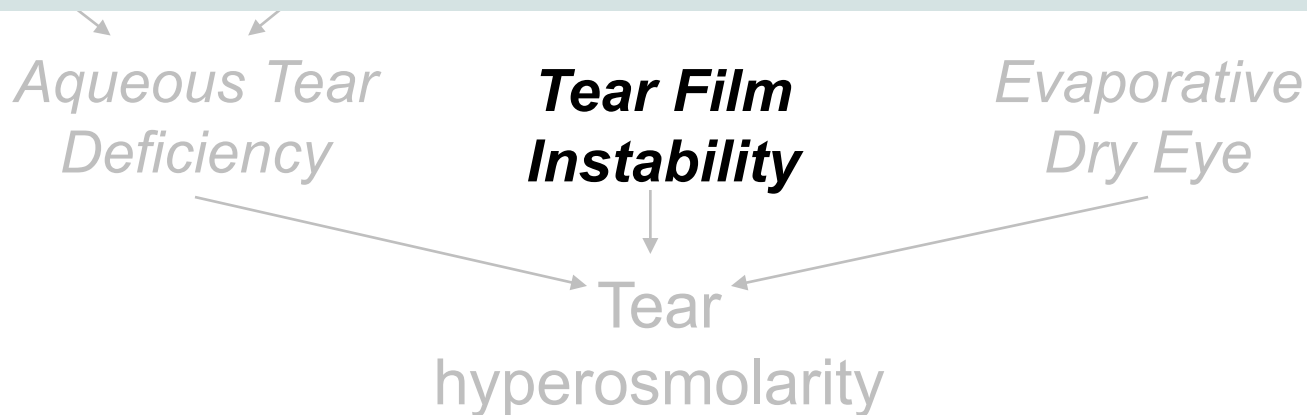
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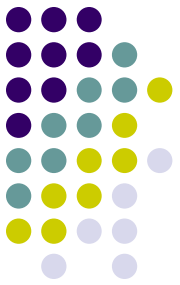
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*A TBUT of less than how long is considered abnormal?*



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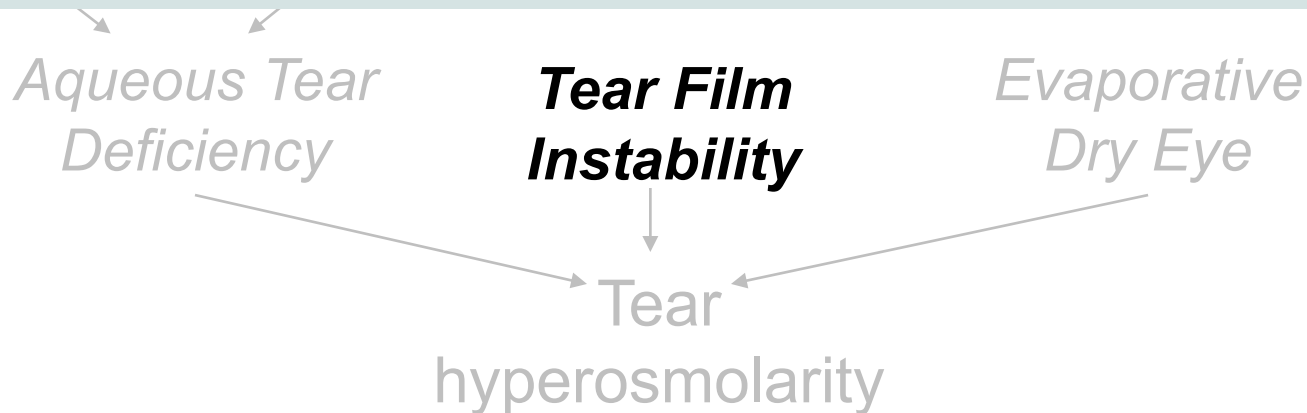
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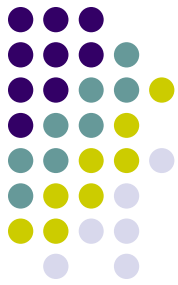
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10 seconds



# Dry Eye Syndrome



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Aqueous Tear  
Deficiency

**Tear Film  
Instability**

Evaporative  
Dry Eye

Tear  
hyperosmolarity



# Dry Eye Syndrome



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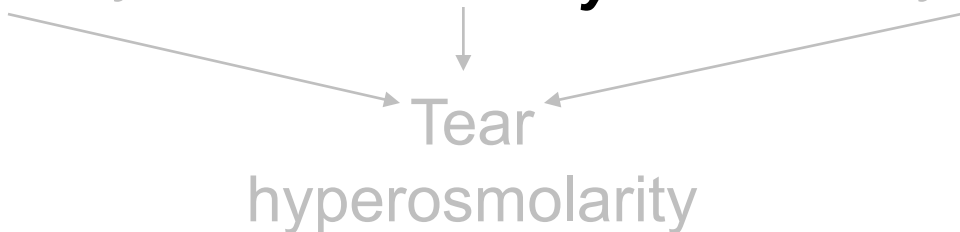
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I assume Fluress drops are the way to go?  
You'd think so, but no. The *Cornea* book states using them is "not recommended" because 1) too much v little fluorescein gets instilled

Aqueous Tear Deficiency

**Tear Film Instability**

Evaporative Dry Eye





# Dry Eye Syndrome



How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?  
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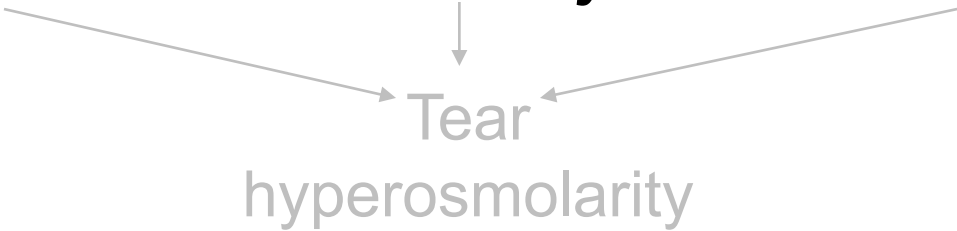
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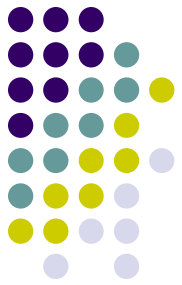
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# Dry Eye Syndrome



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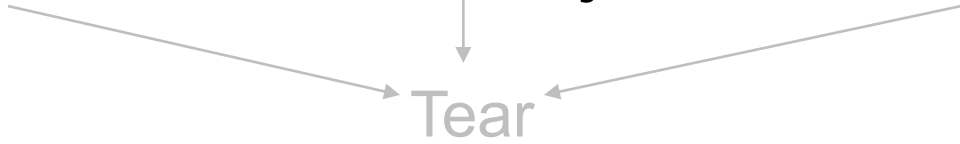
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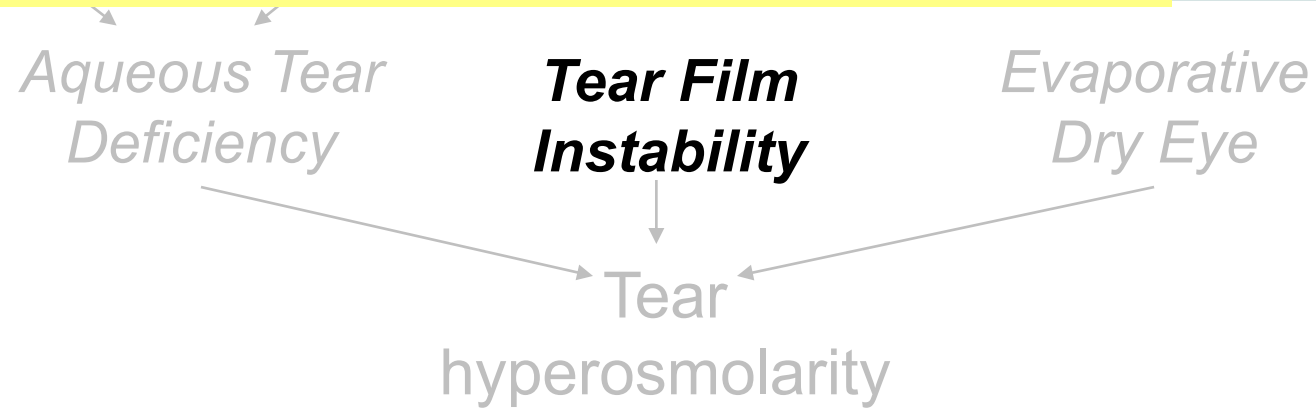
# Dry Eye Syndrome



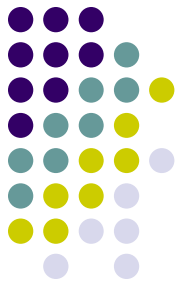
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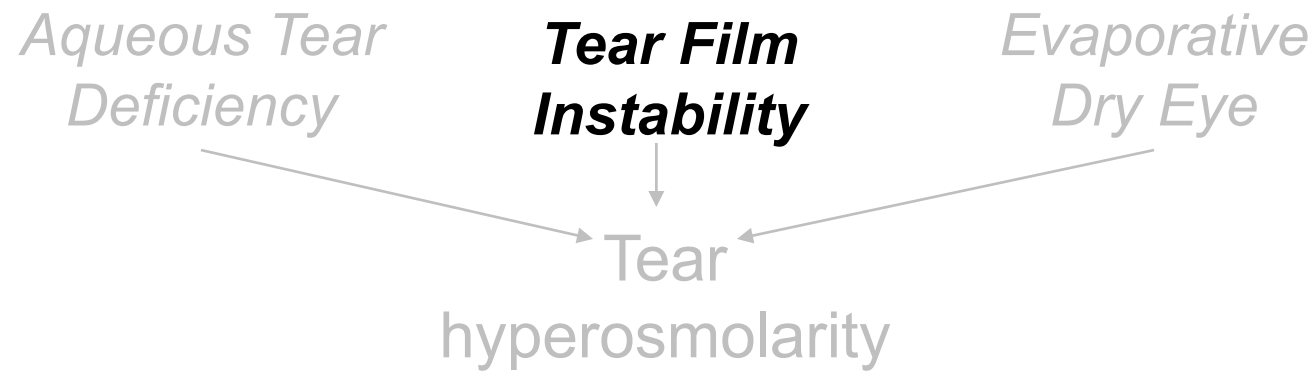
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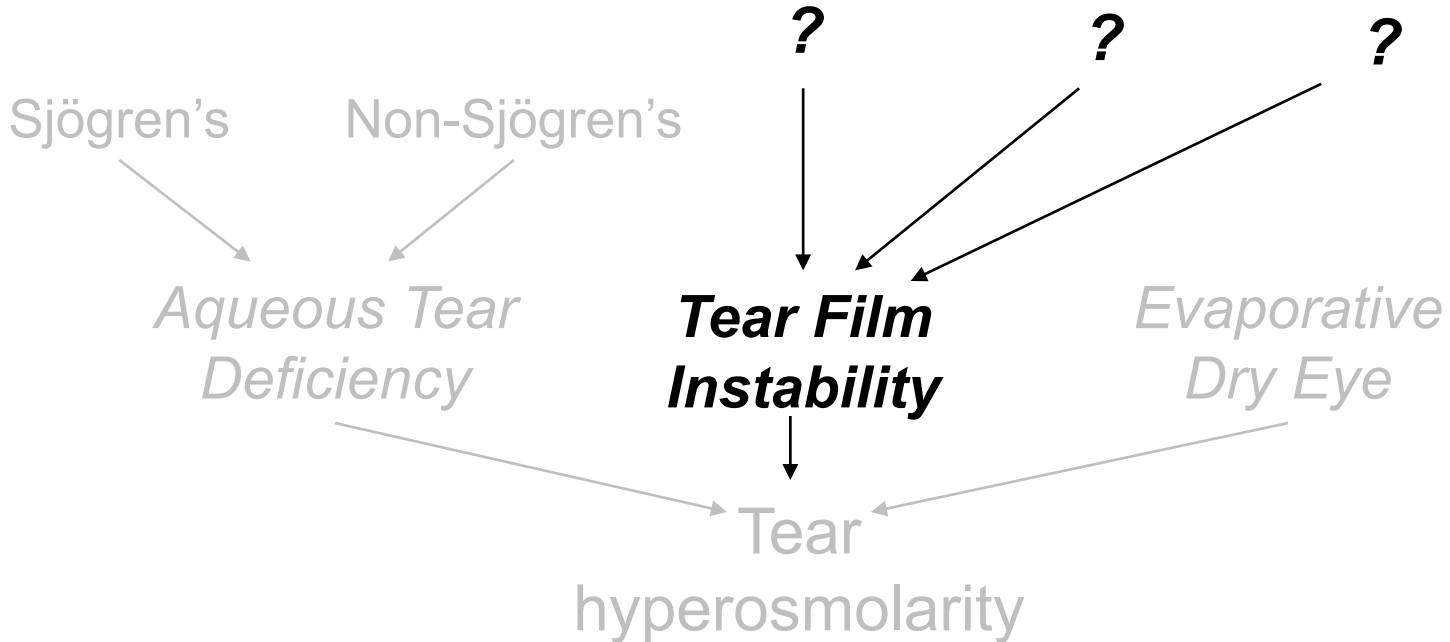
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# Dry Eye Syndrome



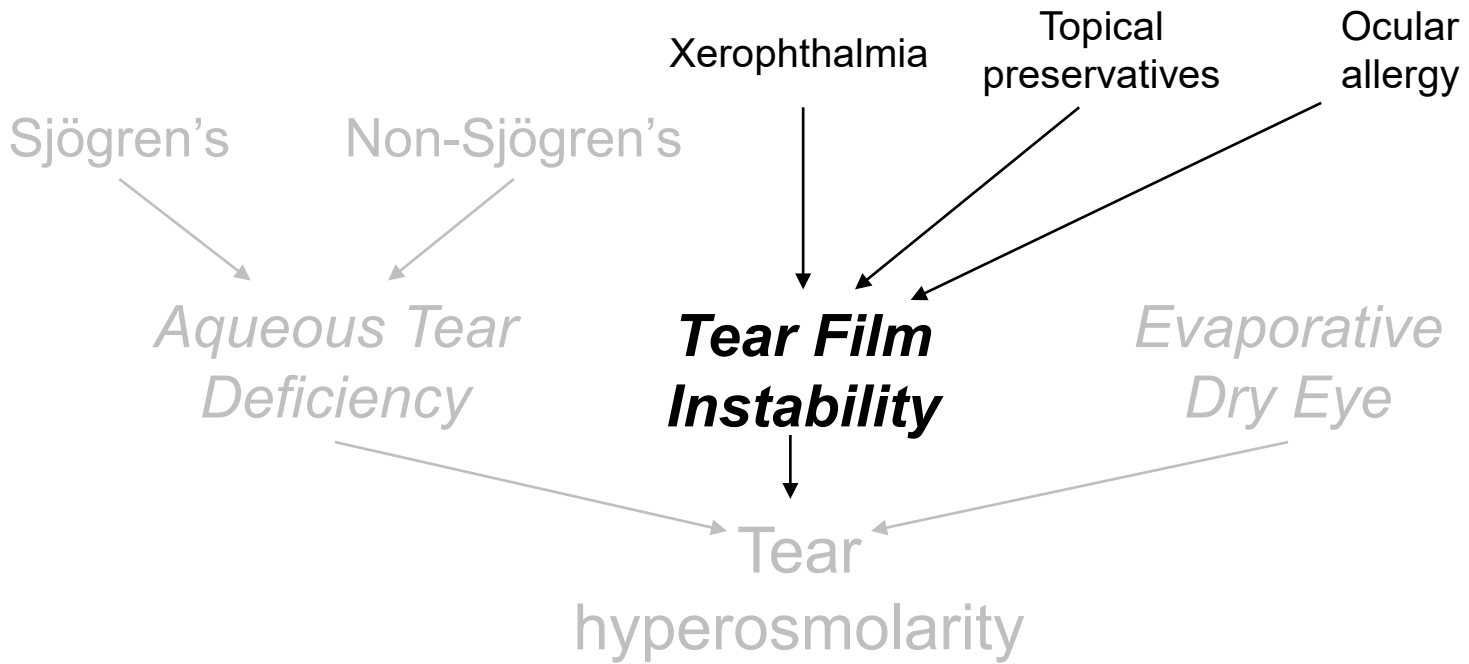
*Three categories of conditions leading to TFI have been identified—what are they?*



# Dry Eye Syndrome



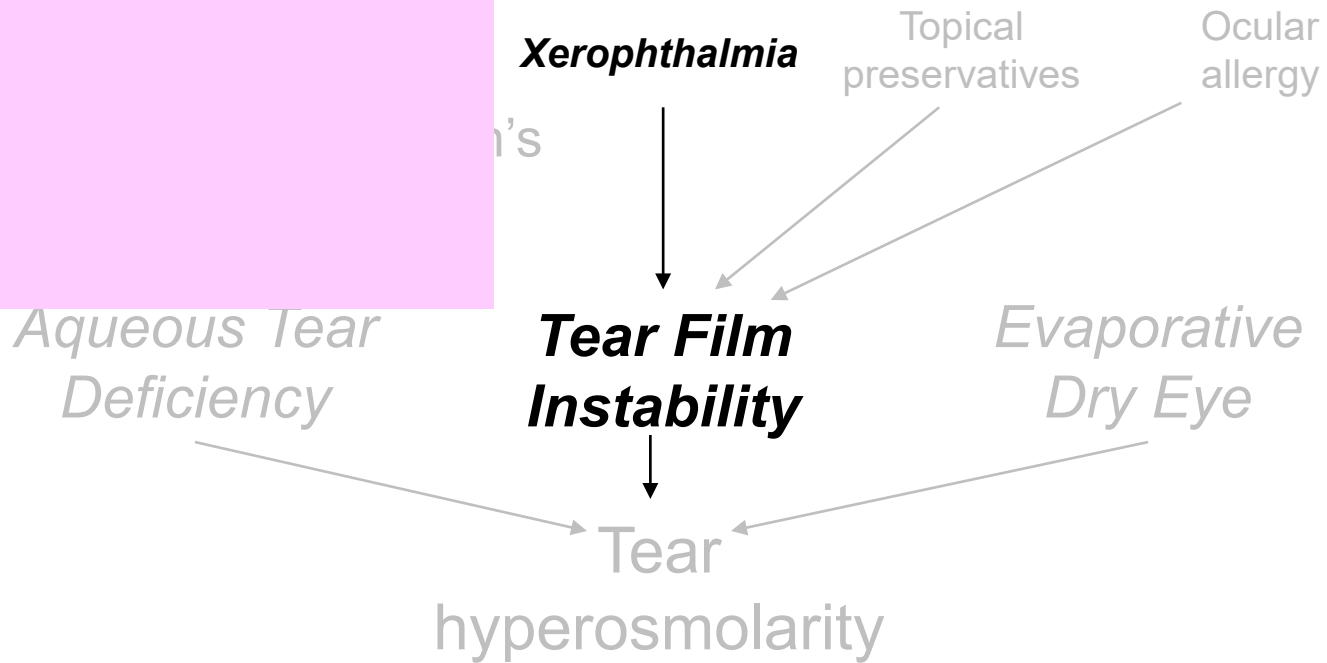
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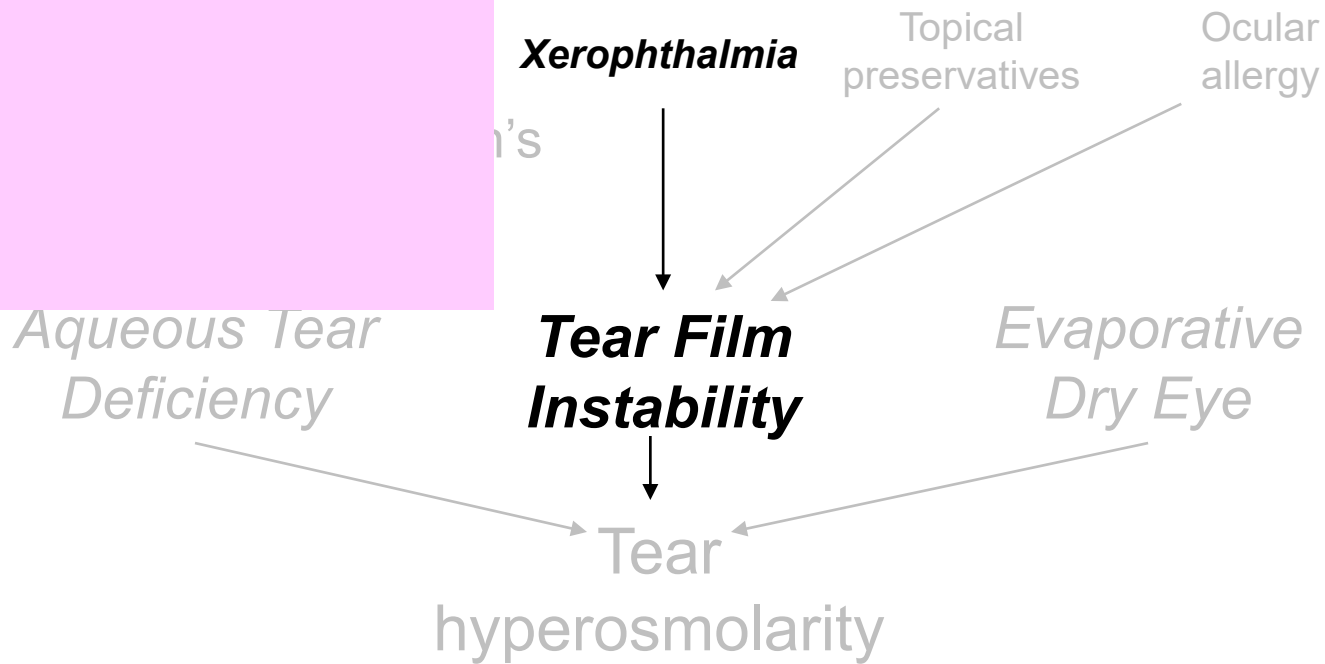
*What nutritional deficiency is the leading cause of xerophthalmia worldwide?*



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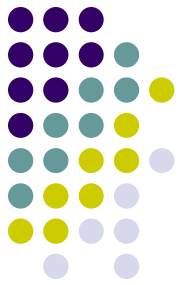


*What nutritional deficiency is the leading cause of xerophthalmia worldwide?*  
Hypovitaminosis A





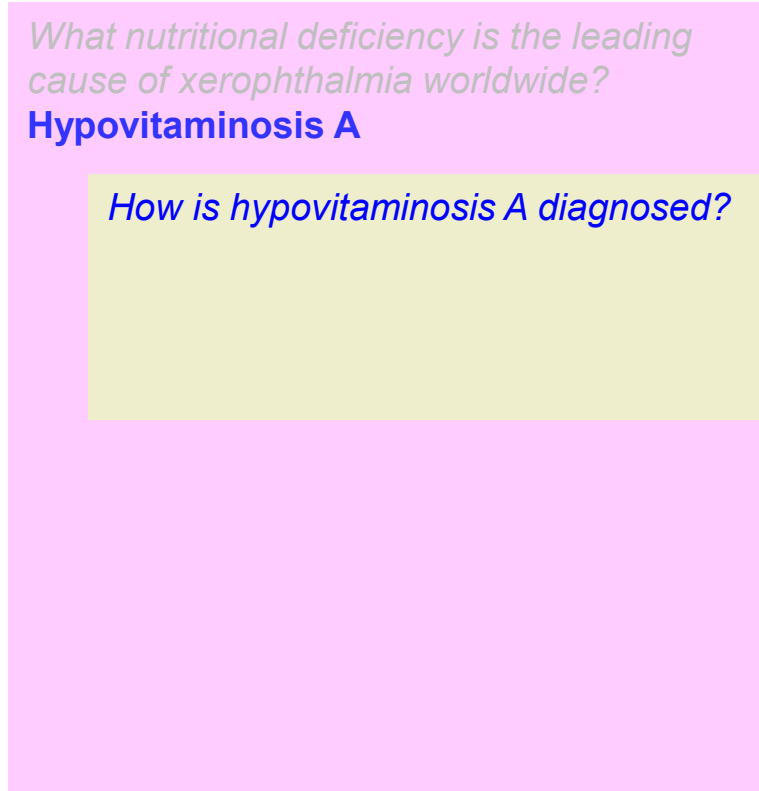
# Dry Eye Syndrome



What nutritional deficiency is the leading cause of xerophthalmia worldwide?

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How is hypovitaminosis A diagnosed?



**Xerophthalmia**

Topical preservatives

Ocular allergy

's

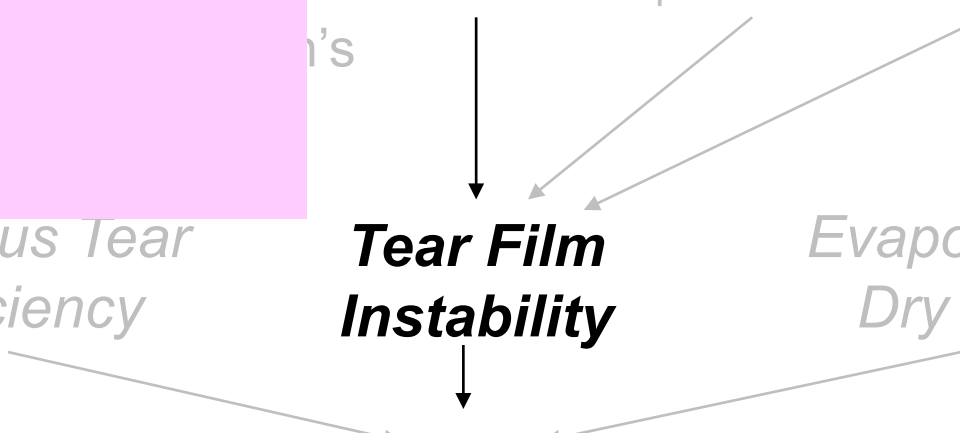
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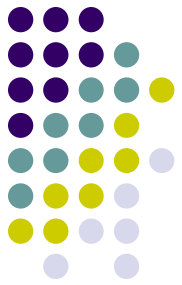
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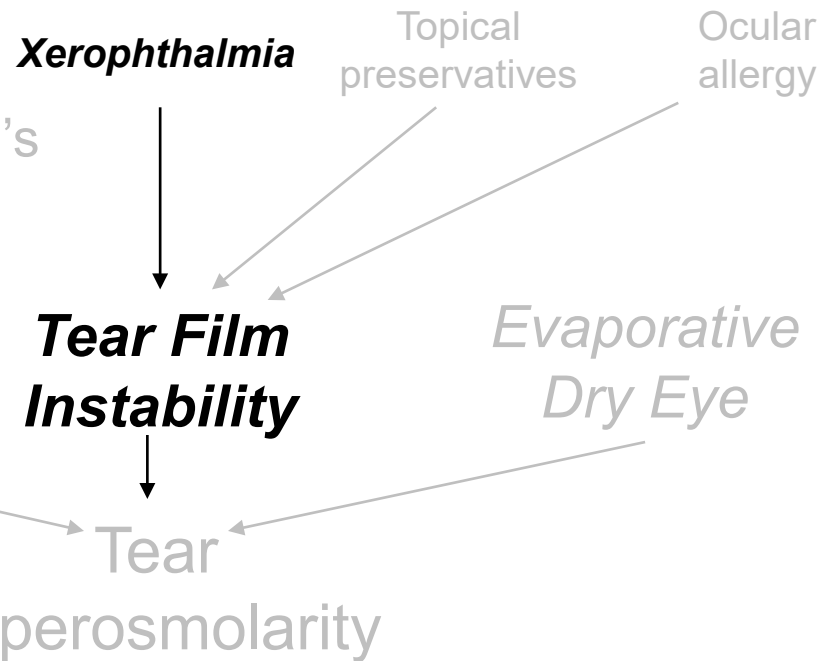
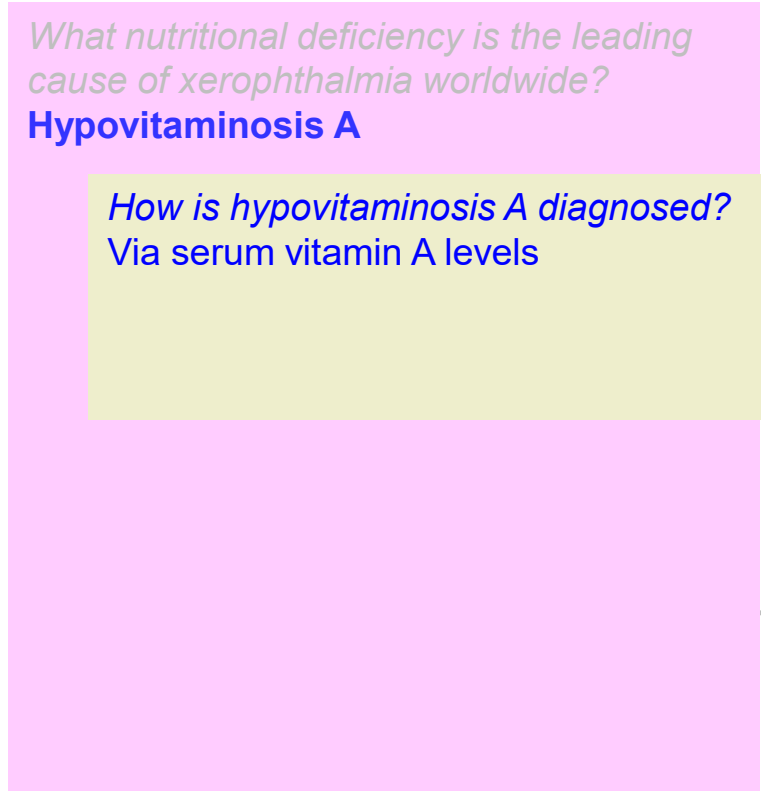
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How is hypovitaminosis A diagnosed?  
Via serum vitamin A levels



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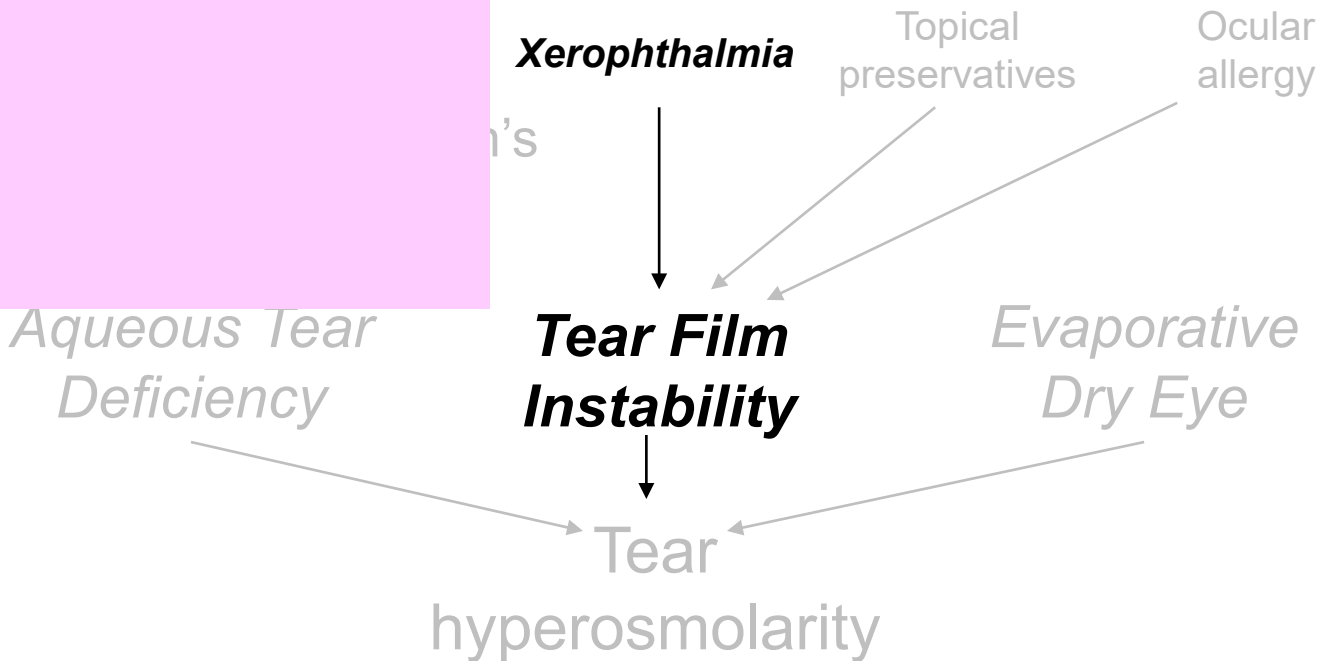
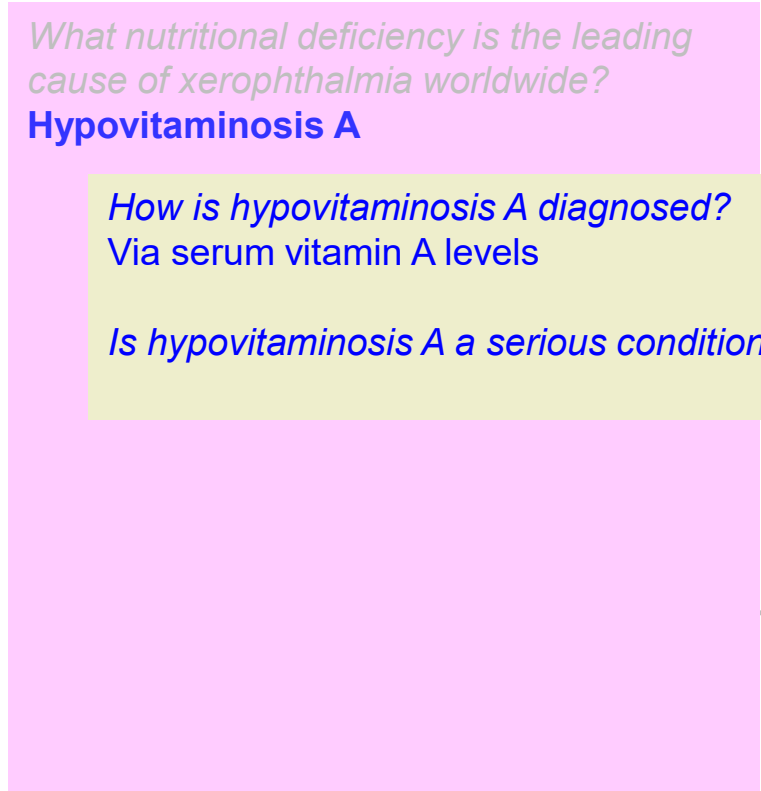
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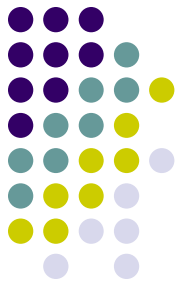
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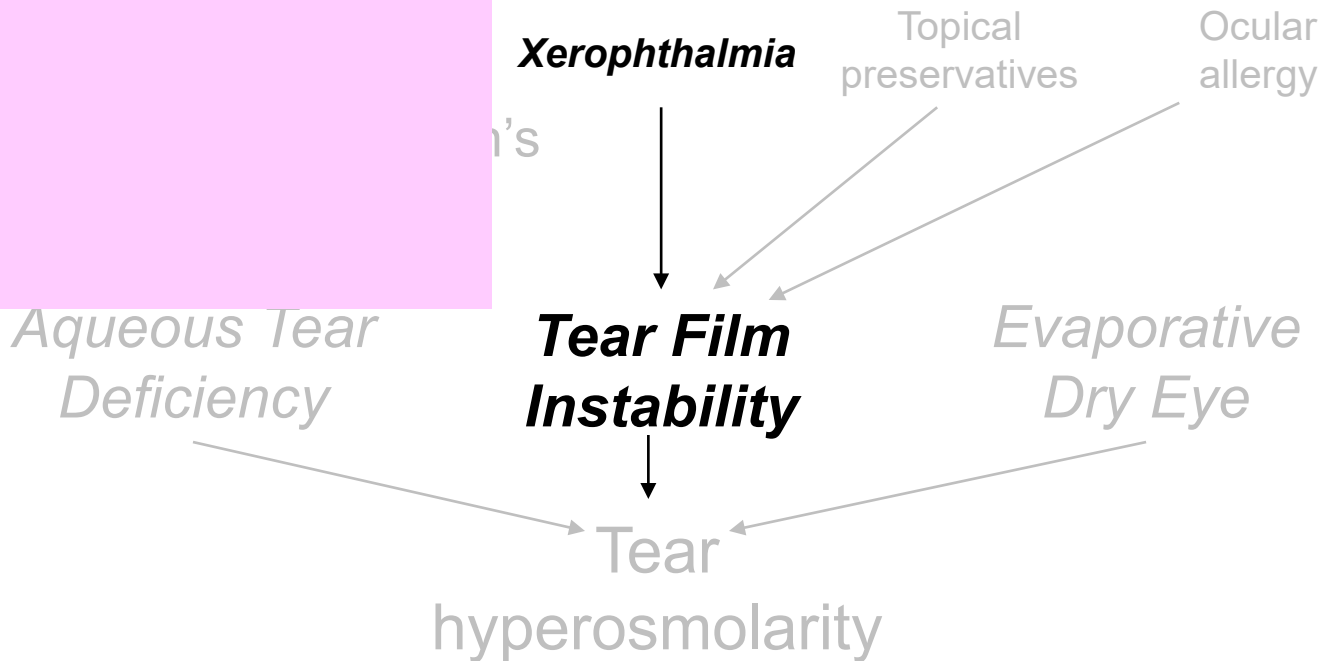
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Is hypovitaminosis A a serious condition?

Yes! The mortality rate is about 50%



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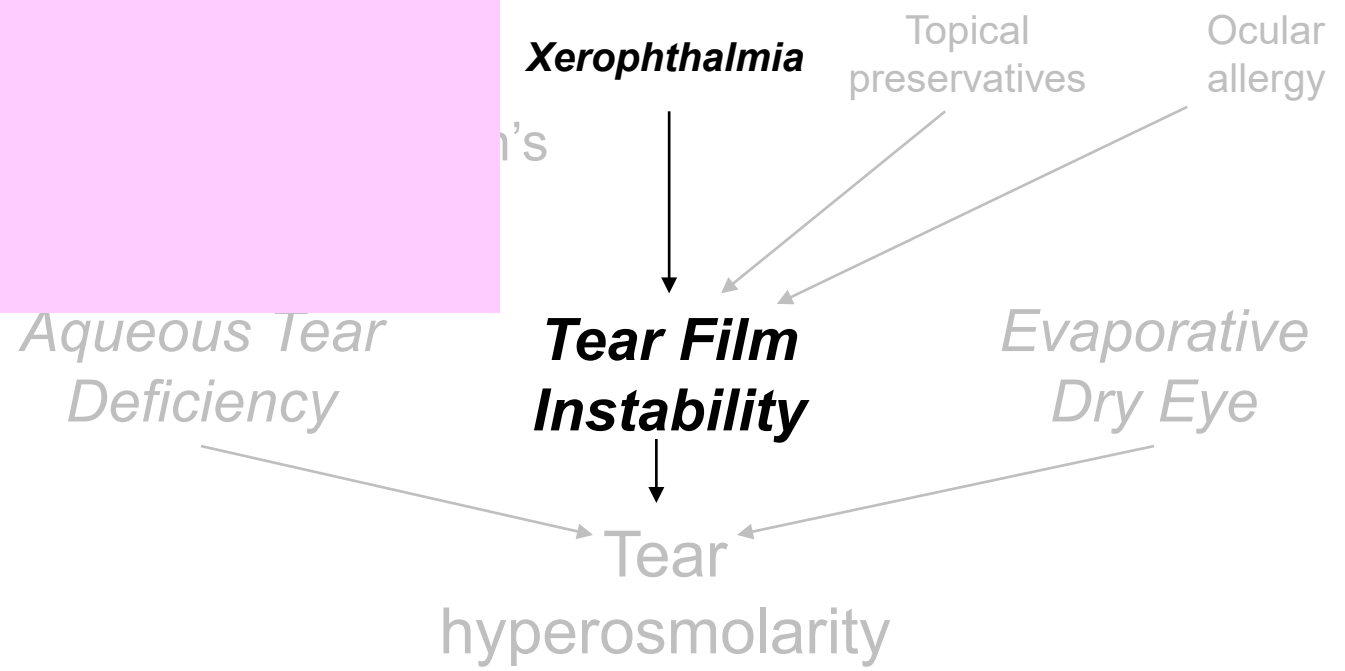


*Xerophthalmia is typically not the first ocular manifestation of hypovitaminosis A. What is?*

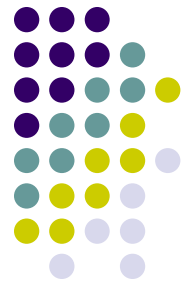
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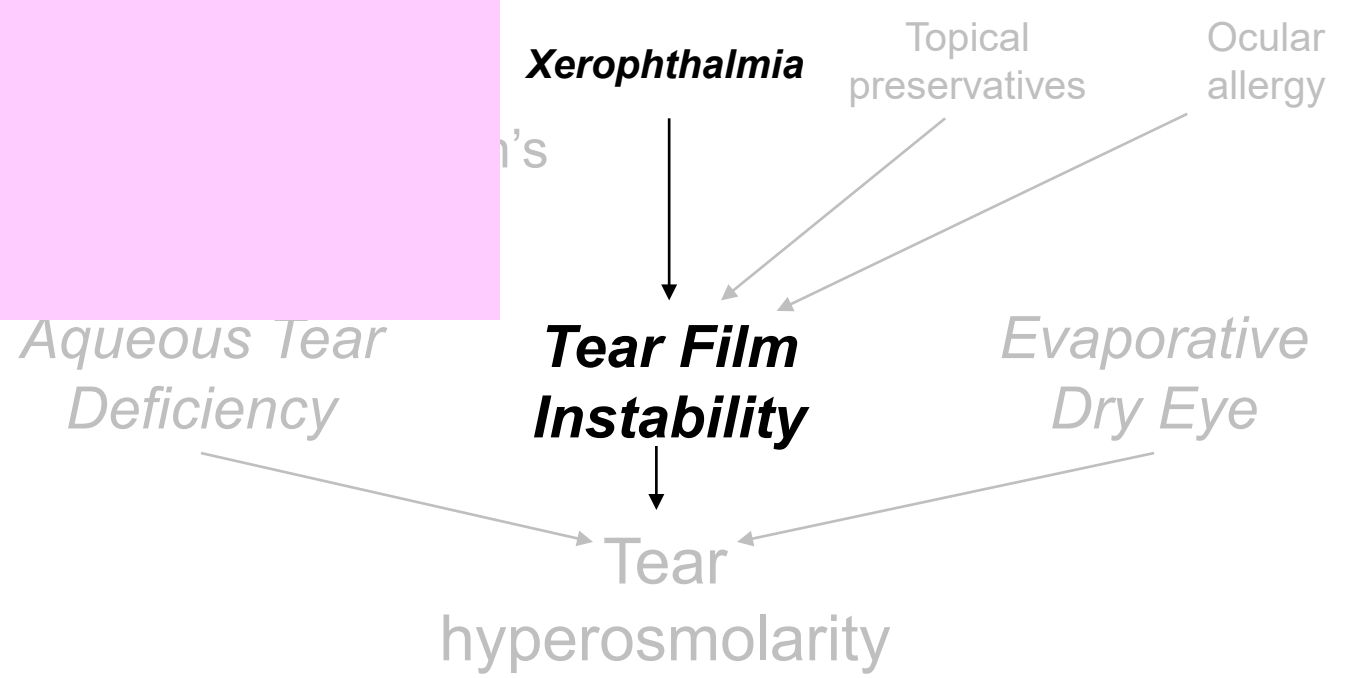


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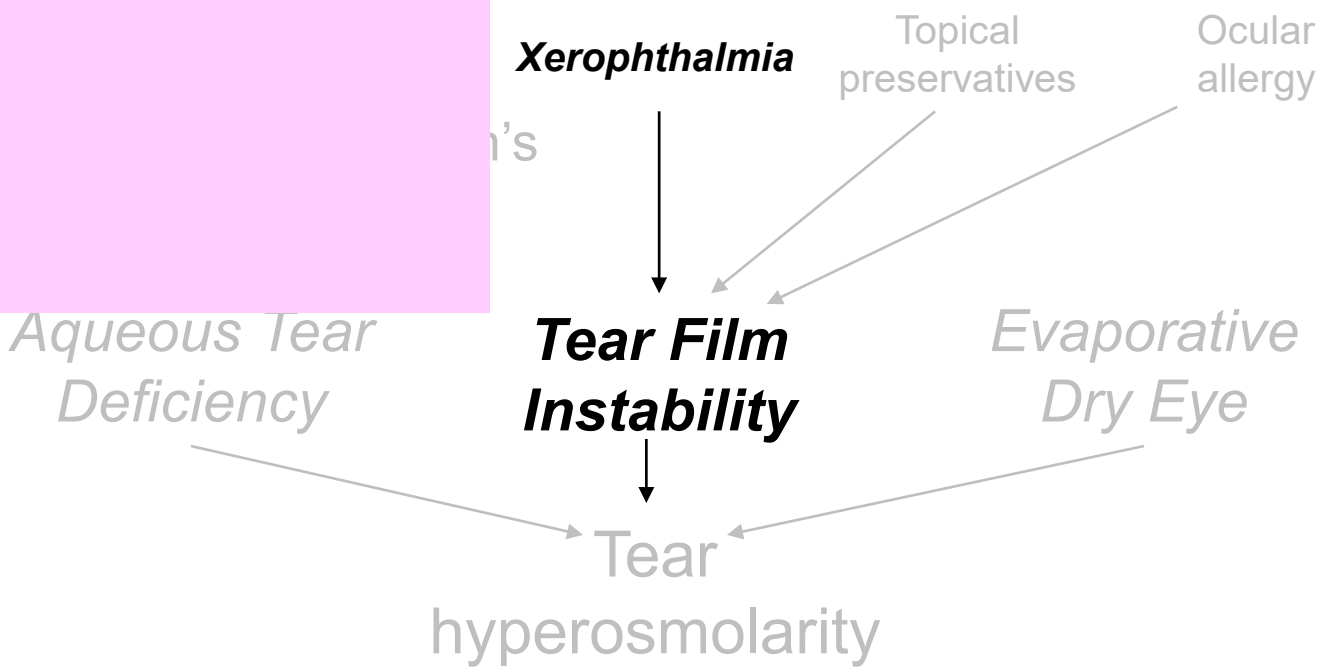


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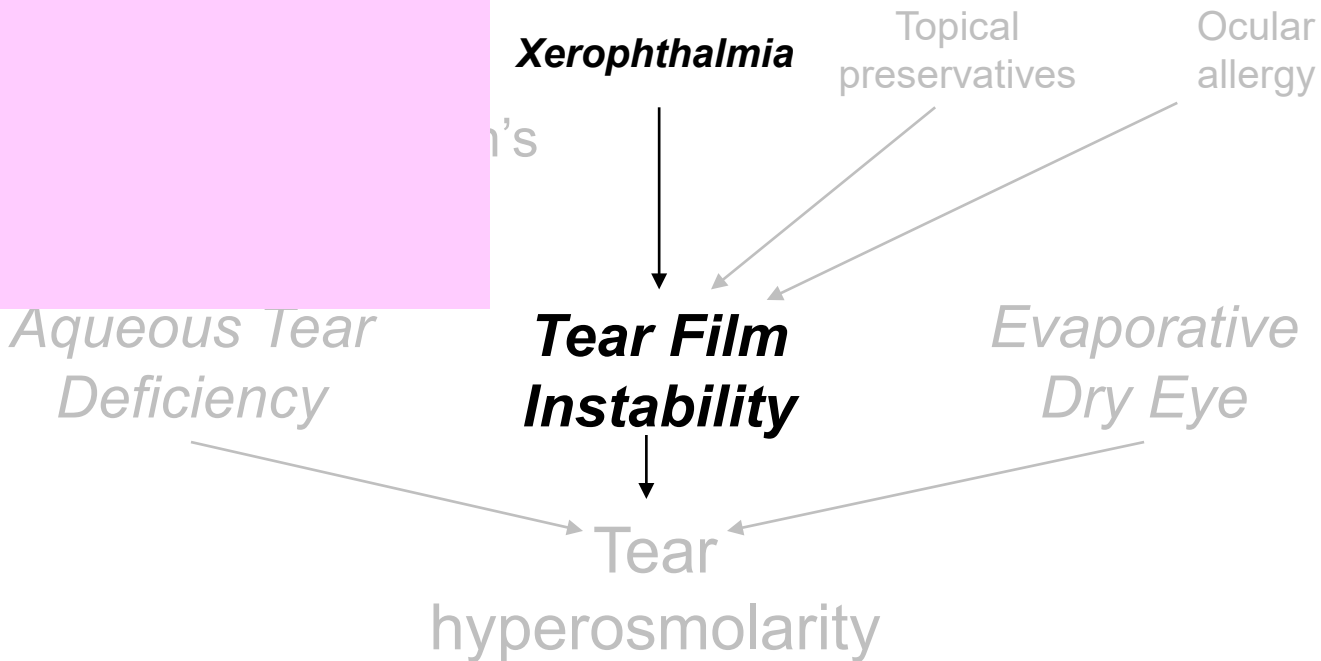


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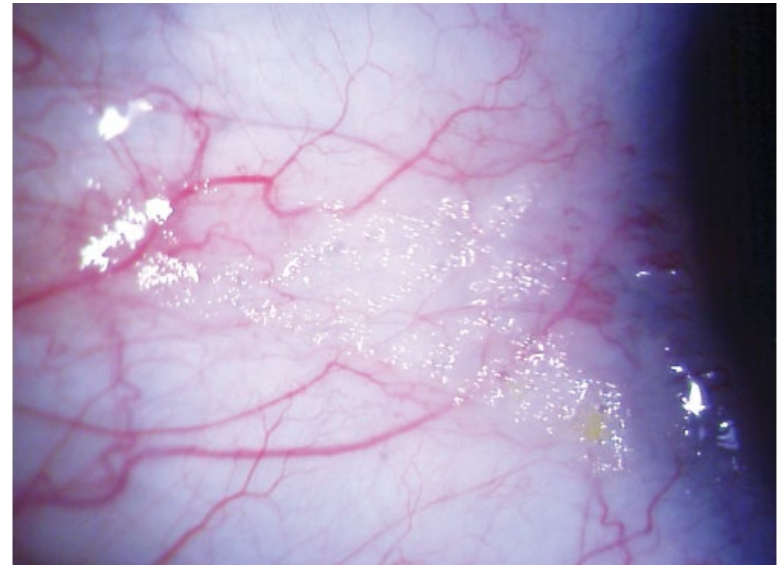
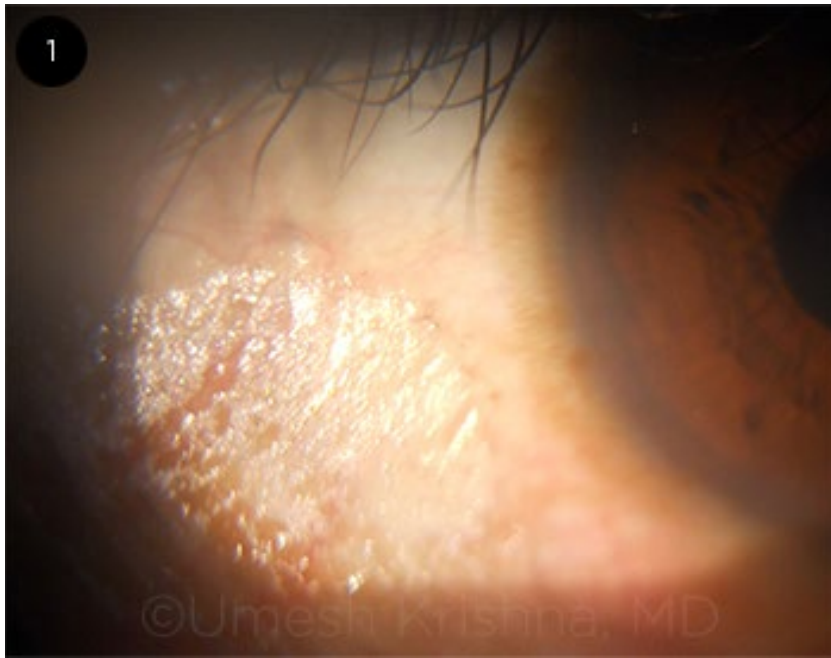
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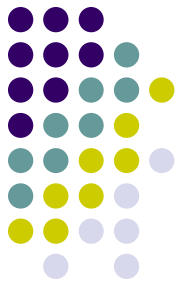


# Dry Eye Syndrome



Bitôt spots: Conj lesion temporal to the cornea, shows typical dry/foamy appearance

# Dry Eye Syndrome



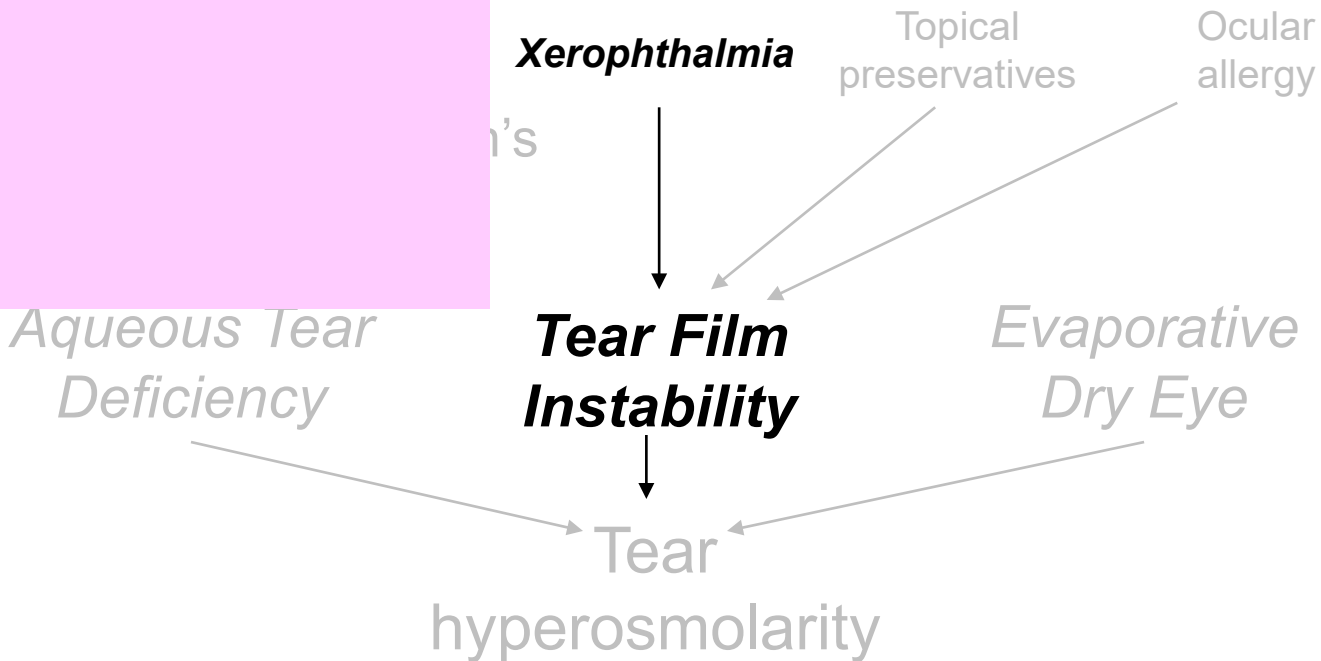
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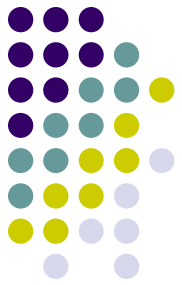
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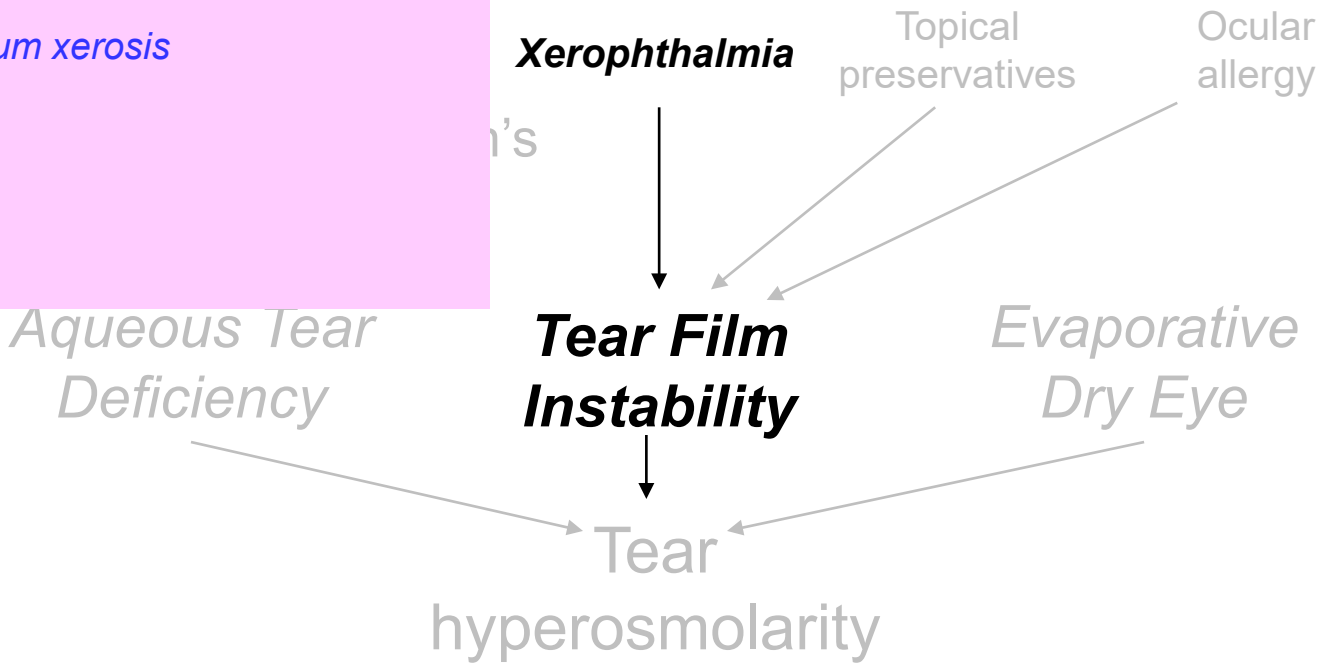
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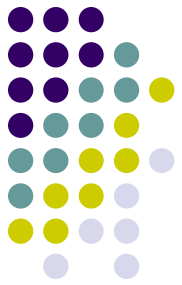
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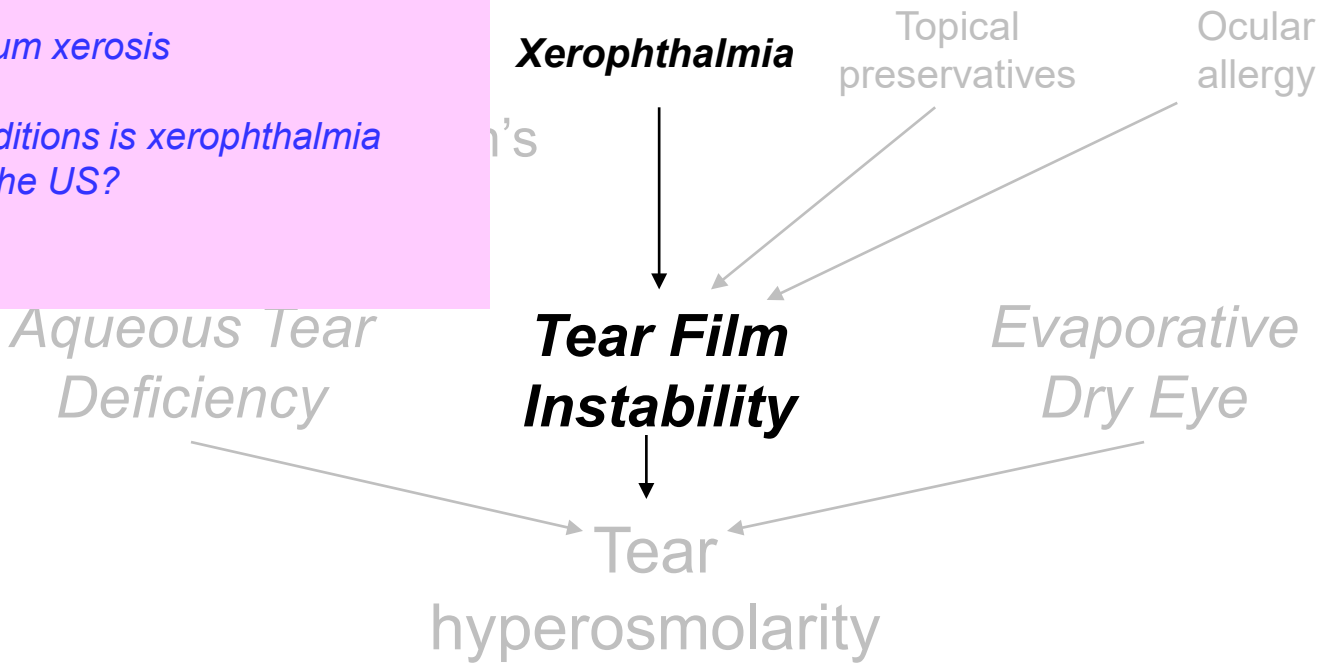
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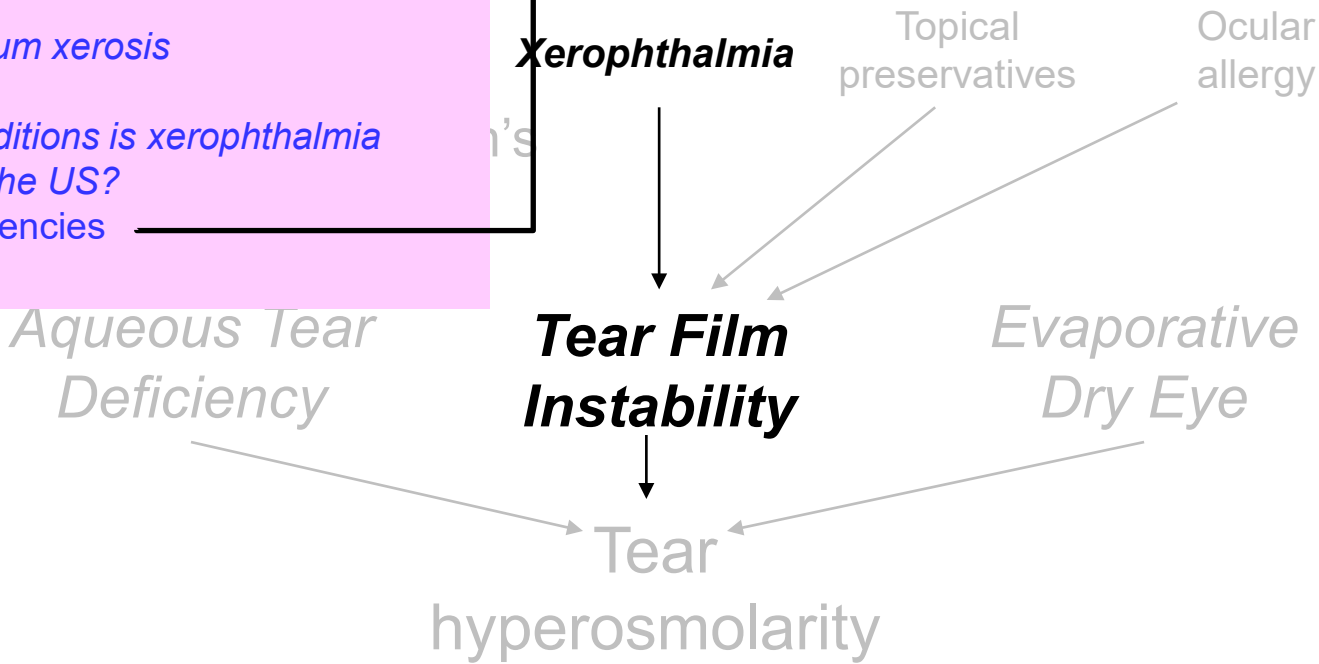


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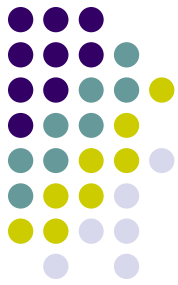
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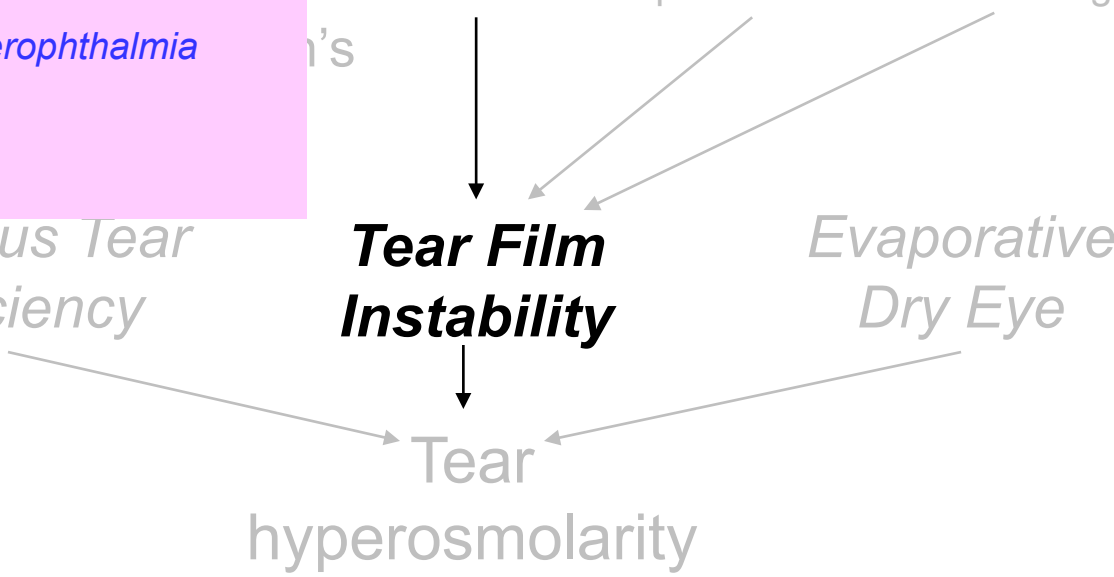
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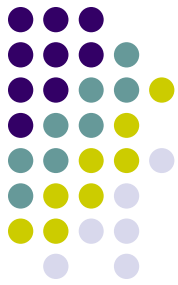
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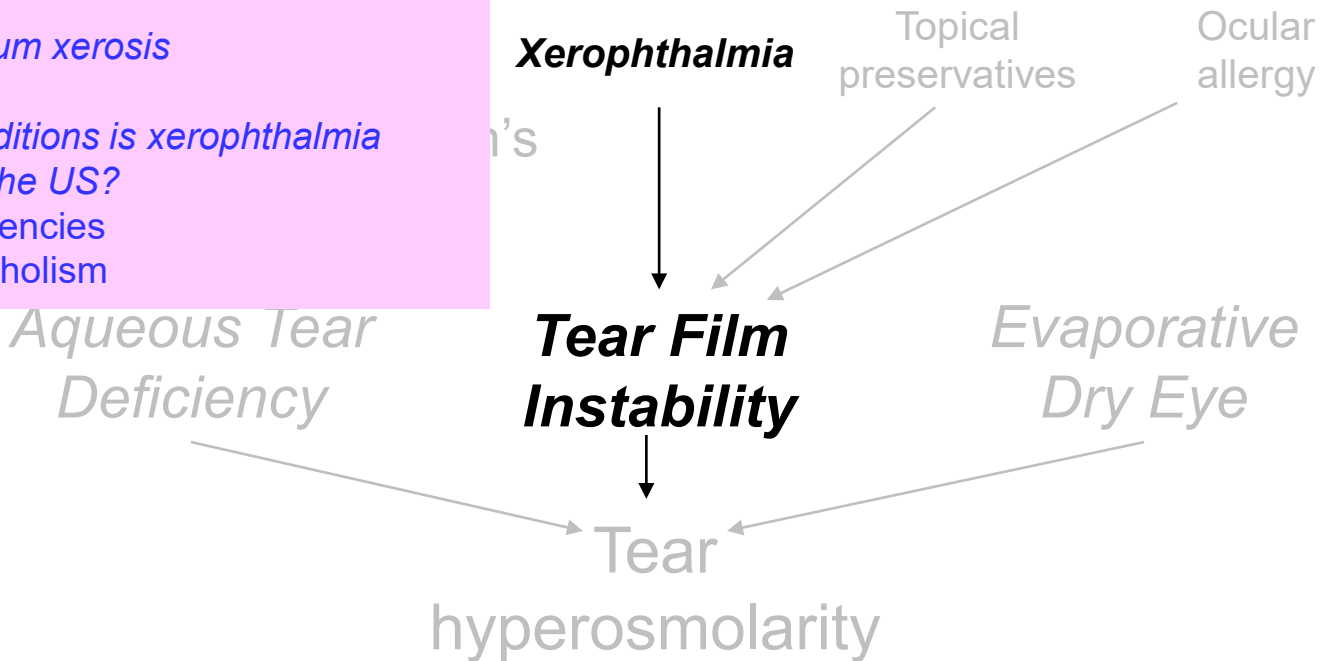
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**Press your xerosis pts on these issues!**

**Xerophthalmia**

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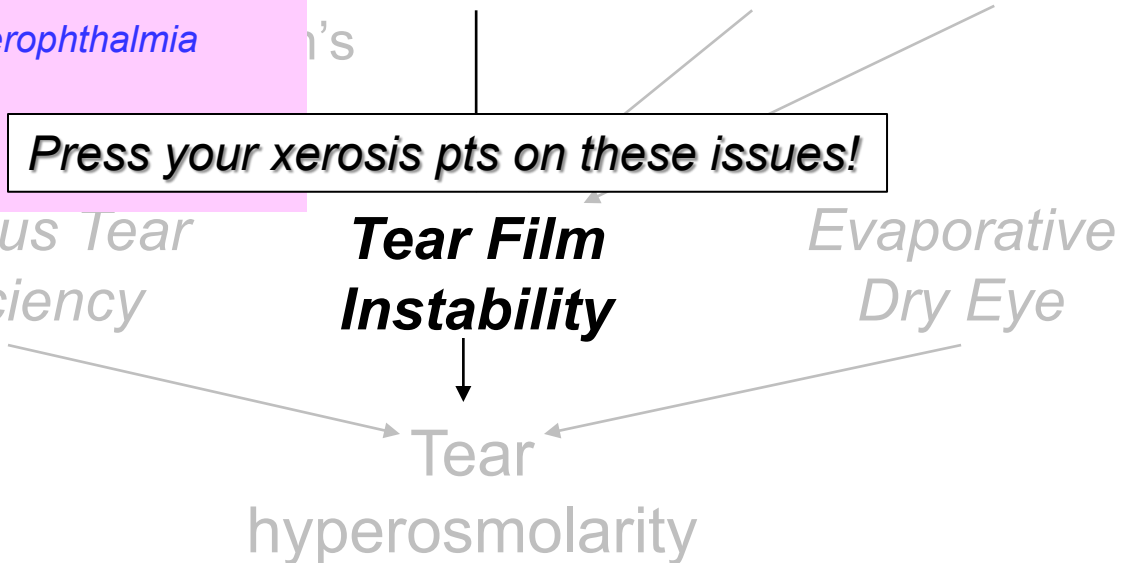
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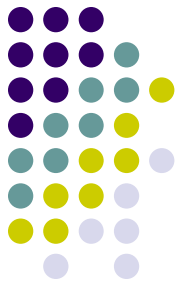
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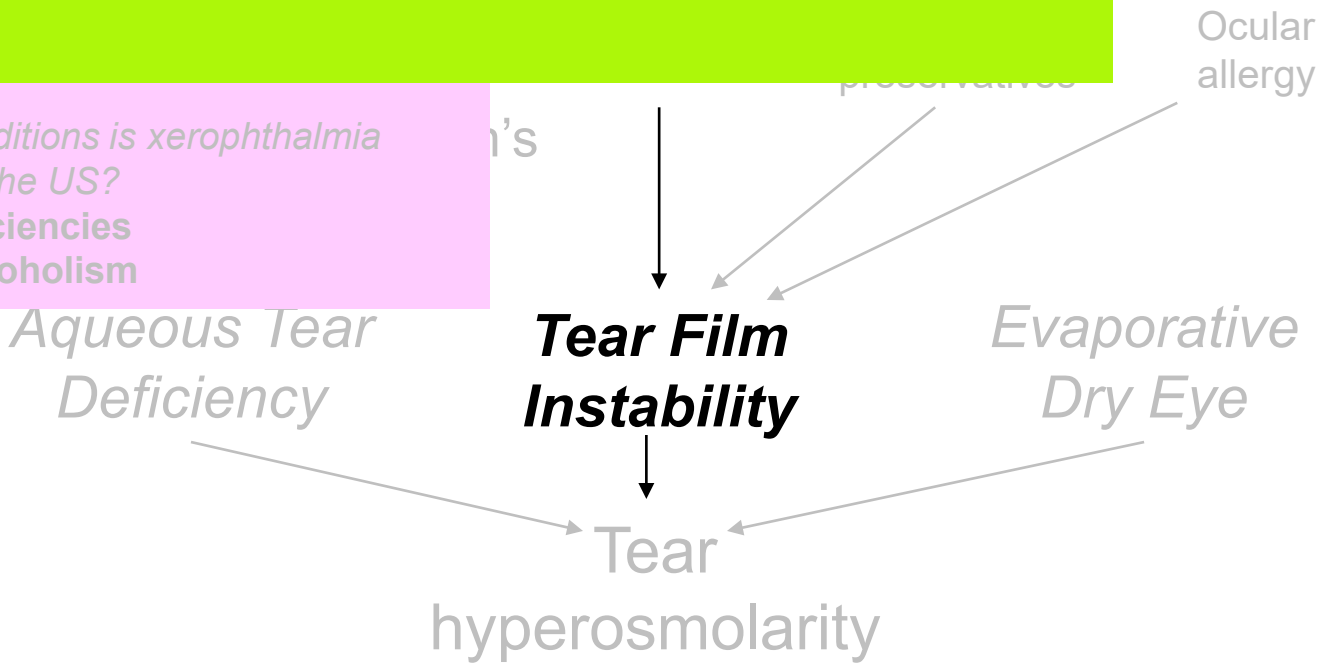
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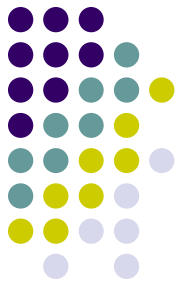
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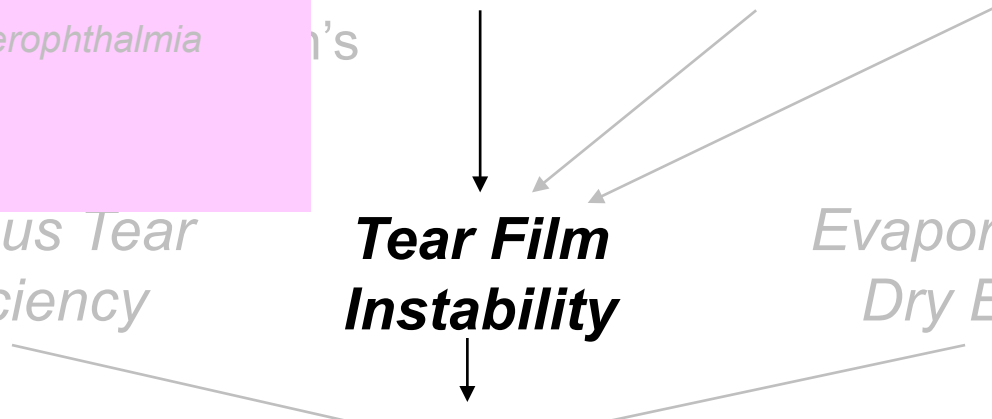
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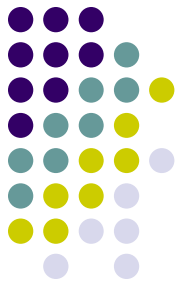
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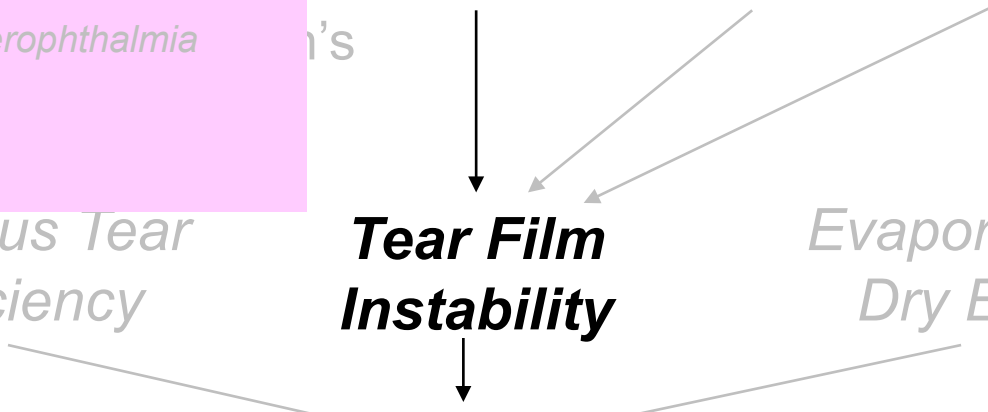
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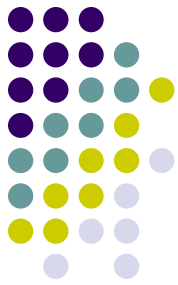
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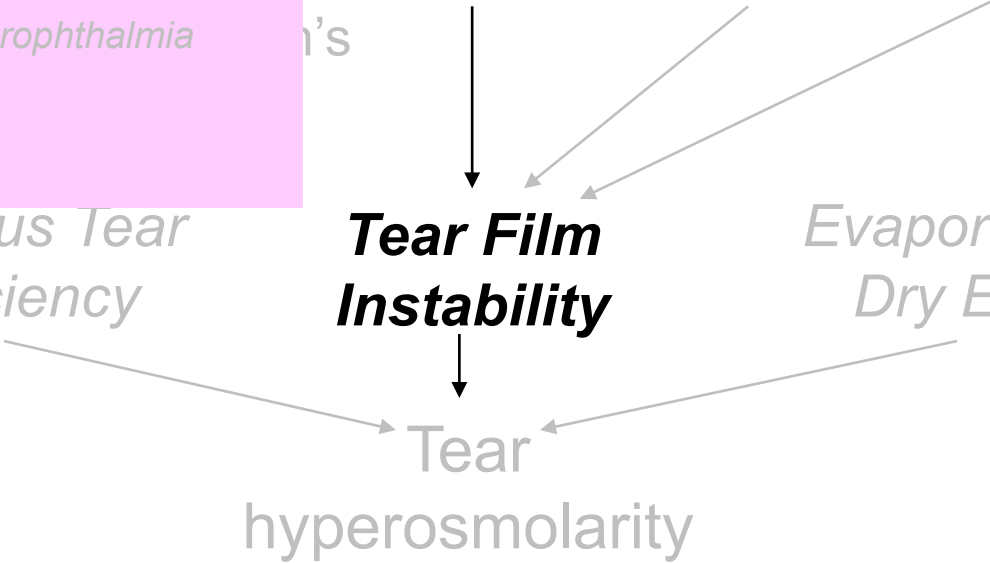
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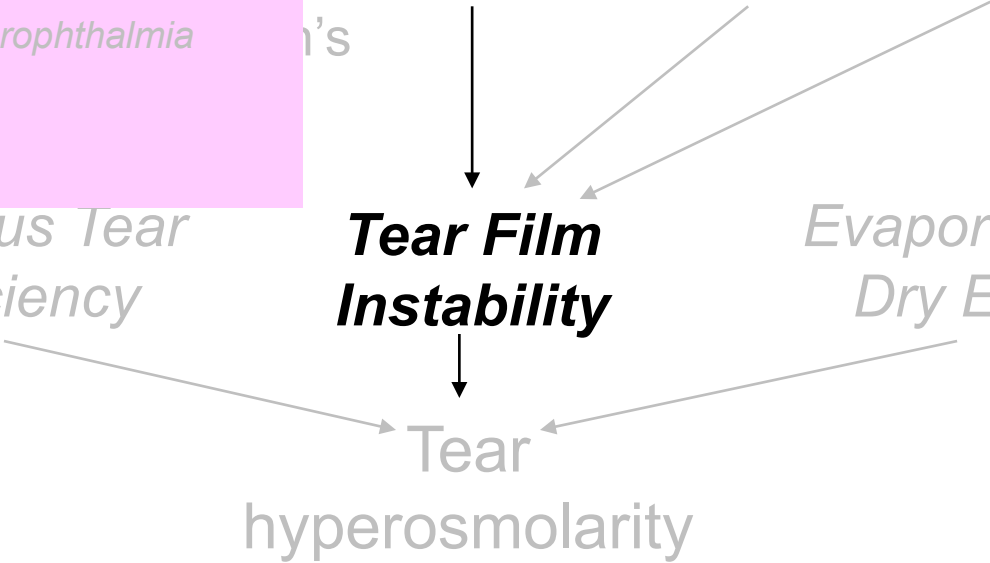
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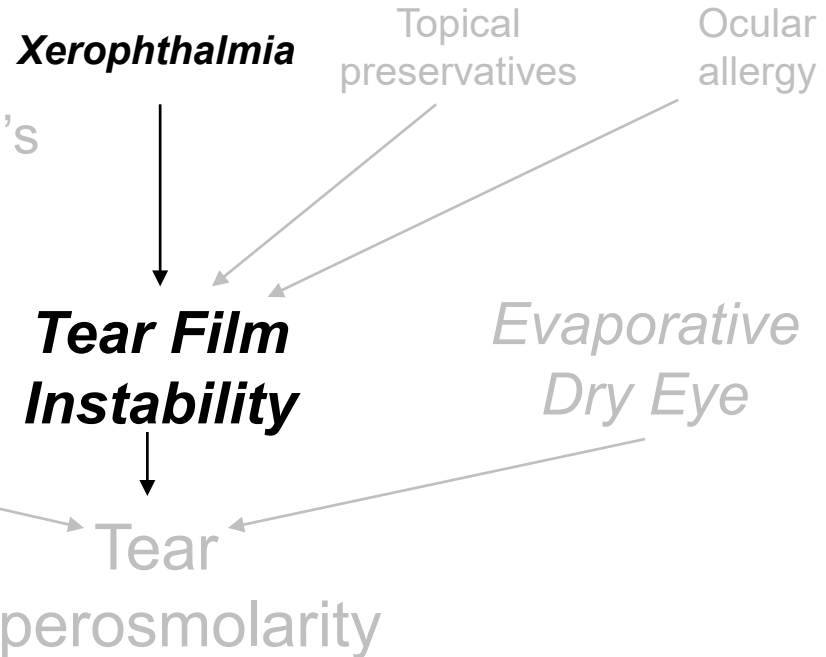
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What foods are rich in vitamin A?

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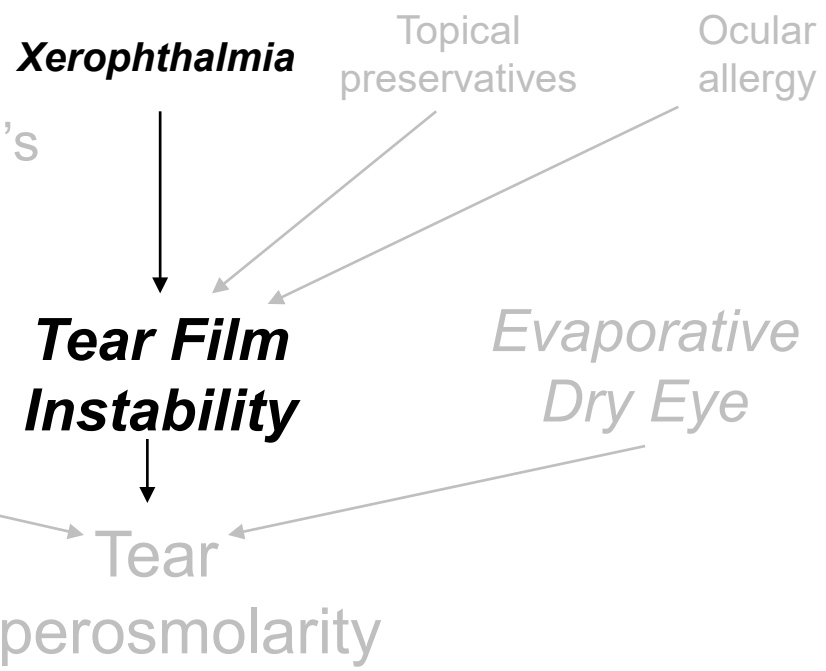
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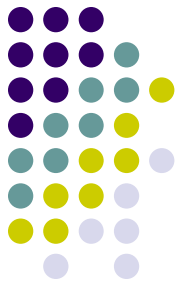
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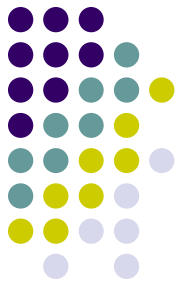
Evaporative Dry Eye

Tear

hyperosmolarity



# Dry Eye Syndrome



What nutritional deficiency is the leading cause of xerophthalmia worldwide?

Hypovitaminosis A

Hypovitaminosis A xerosis of the ocular surface produces what classic sign?

**Bitôt spot**—a foamy, white/gray area on the interpalpebral conjunctiva

What bacteria is implicated in Bitôt spot formation?

*Corynebacterium xerosis*

With what conditions is xerophthalmia associated in the US?

--Dietary deficiencies

--Chronic alcoholism

What foods are rich in vitamin A?

--Organ meat, especially liver

--Oily fish

--Carrots

--Dark green leafy veggies

**Xerophthalmia**

Topical preservatives

Ocular allergy

**Tear Film Instability**

Evaporative Dry Eye

Tear

hyperosmolarity



# Dry Eye Syndrome



What nutritional deficiency is the leading cause of xerophthalmia worldwide?

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**Bitôt spot**—a foamy, white, foamy patch on the interpalpebral conjunctiva

What bacteria is implicated in the formation of Bitôt spots?

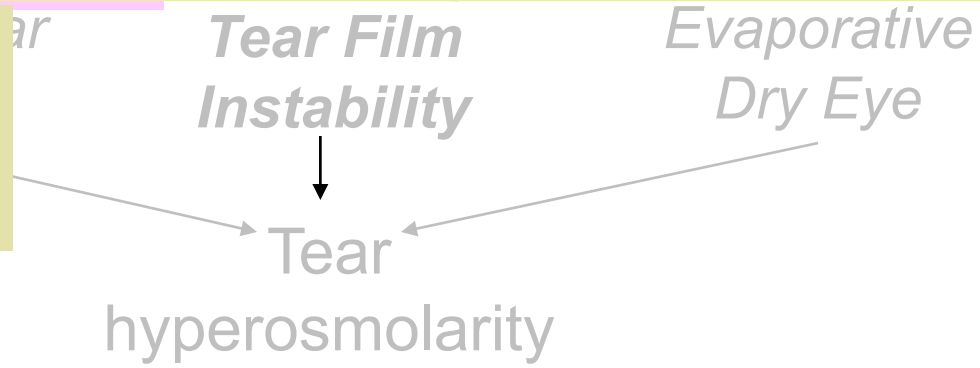
*Corynebacterium xerosis*

With what conditions is xerophthalmia associated in the US?

- Dietary deficiencies**
- Chronic alcoholism**

*Is hypervitaminosis A a thing, ie, a clinically important condition?*

- What foods are rich in vitamin A?
- Organ meat, especially liver
  - Oily fish
  - Carrots
  - Dark green leafy veggies





# Dry Eye Syndrome



What nutritional deficiency is the leading cause of xerophthalmia worldwide?

Hypovitaminosis A

Hypovitaminosis A xerosis of the ocular surface produces what classic sign?

**Bitôt spot**—a foamy, white, foamy, white patches on the interpalpebral conjunctiva

**Is hypervitaminosis A a thing, ie, a clinically important condition?**

It is indeed

What bacteria is implicated in the formation of Bitôt spots?

*Corynebacterium xerosis*

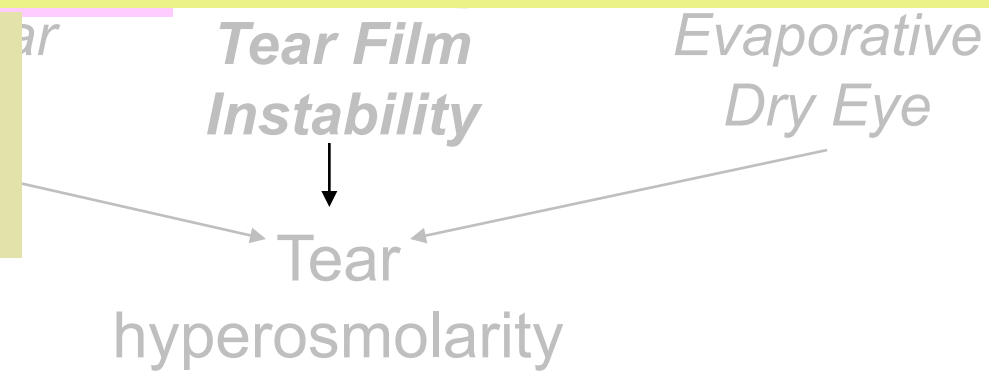
*There is a condition of significant ophthalmic consequence—one with which you are likely familiar—that has a strong association with hypervitaminosis A. What is it?*

With what conditions is xerophthalmia associated in the US?

- Dietary deficiencies
- Chronic alcoholism

What foods are rich in vitamin A?

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# Dry Eye Syndrome



What nutritional deficiency is the leading cause of xerophthalmia worldwide?

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There is a condition of significant ophthalmic consequence—one with which you are likely familiar—that has a strong association with hypervitaminosis A. What is it?

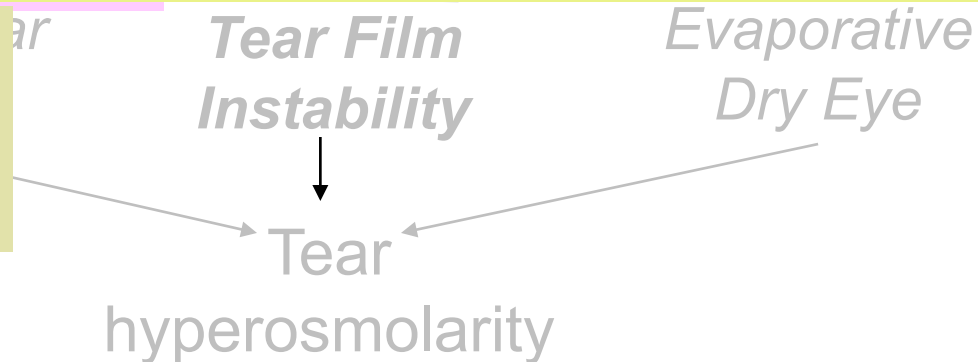
Idiopathic\* intracranial hypertension (aka two words)

With what conditions is xerophthalmia associated in the US?

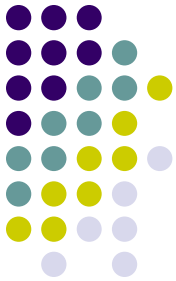
- Dietary deficiencies
- Chronic alcoholism

What foods are rich in vitamin A?

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- Oily fish
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- Dark green leafy veggies



# Dry Eye Syndrome



*What nutritional deficiency is the leading cause of xerophthalmia worldwide?*  
 Hypovitaminosis A

*Hypovitaminosis A xerosis of the ocular surface produces what classic sign?*  
**Bitôt spot**—a foamy, white, foamy lesion of the interpalpebral conjunctiva

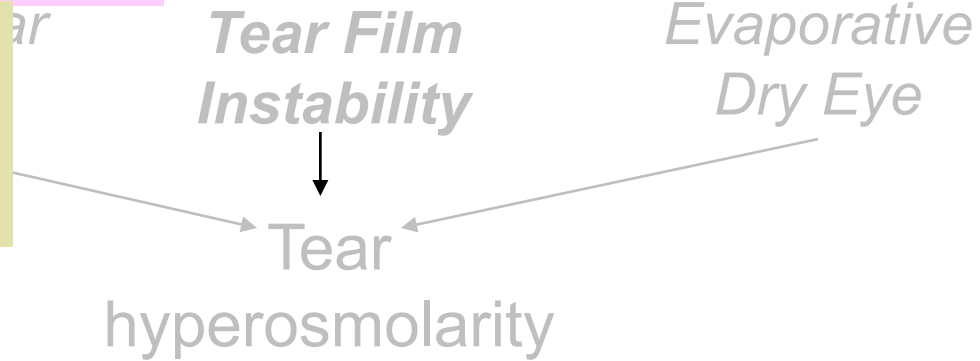
*What bacteria is implicated in conjunctival xerosis formation?*  
*Corynebacterium xerosis*

*With what conditions is xerophthalmia associated in the US?*  
 --Dietary deficiencies  
 --Chronic alcoholism

*Is hypervitaminosis A a thing, ie, a clinically important condition?*  
 It is indeed

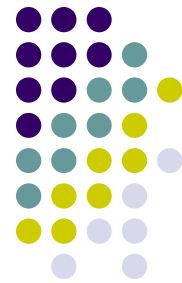
*There is a condition of significant ophthalmic consequence—one with which you are likely familiar—that has a strong association with hypervitaminosis A. What is it?*  
 Idiopathic\* intracranial hypertension (aka *pseudotumor cerebri*)

*What foods are rich in vitamin A?*  
 --Organ meat, especially liver  
 --Oily fish  
 --Carrots  
 --Dark green leafy veggies





# Dry Eye Syndrome



What nutritional deficiency is the leading cause of xerophthalmia worldwide?

Hypovitaminosis A

Hypovitaminosis A xerosis of the ocular surface produces what classic sign?

**Bitôt spot**—a foamy, white, foamy, white, the interpalpebral conjunctiva

**Is hypervitaminosis A a thing, ie, a clinically important condition?**

It is indeed

What bacteria is implicated in conjunctival squamous metaplasia and squamous metaplasia formation?

*Corynebacterium xerosis*

*There is a condition of significant ophthalmic consequence—one with which you are likely familiar—that has a strong association with hypervitaminosis A. What is it?*

Idiopathic\* intracranial hypertension (aka *pseudotumor cerebri*)

With what conditions is xerophthalmia associated in the US?

--Dietary deficiencies

--Chronic alcoholism

*There is a classic (albeit far-fetched) dietary scenario associated with the development of pseudotumor cerebri—what is it?*

What foods are rich in vitamin A?

--Organ meat, especially liver

--Oily fish

--Carrots

--Dark green leafy veggies

*Tear Film Instability*

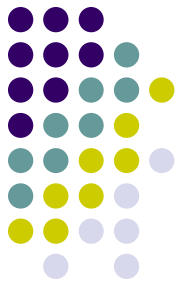
*Evaporative Dry Eye*

Tear

hyperosmolarity



# Dry Eye Syndrome



*What nutritional deficiency is the leading cause of xerophthalmia worldwide?*

Hypovitaminosis A

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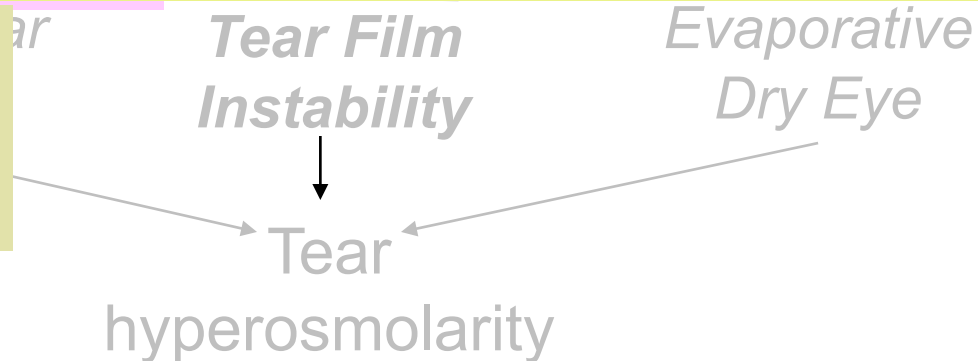
- Dietary deficiencies
- Chronic alcoholism

*There is a classic (albeit far-fetched) dietary scenario associated with the development of pseudotumor cerebri—what is it?*

Consumption of three words

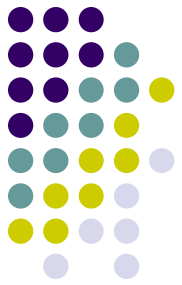
*What foods are rich in vitamin A?*

- Organ meat, especially liver
- Oily fish
- Carrots
- Dark green leafy veggies

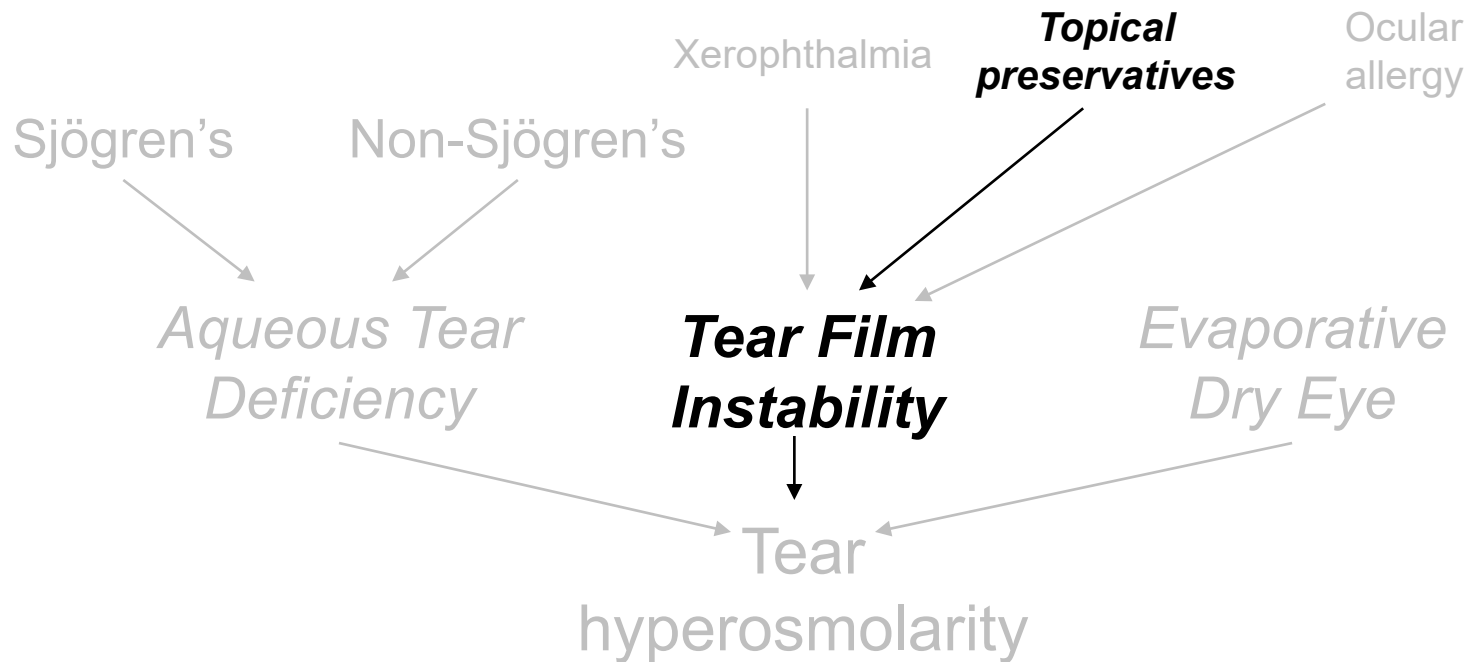




# Dry Eye Syndrome



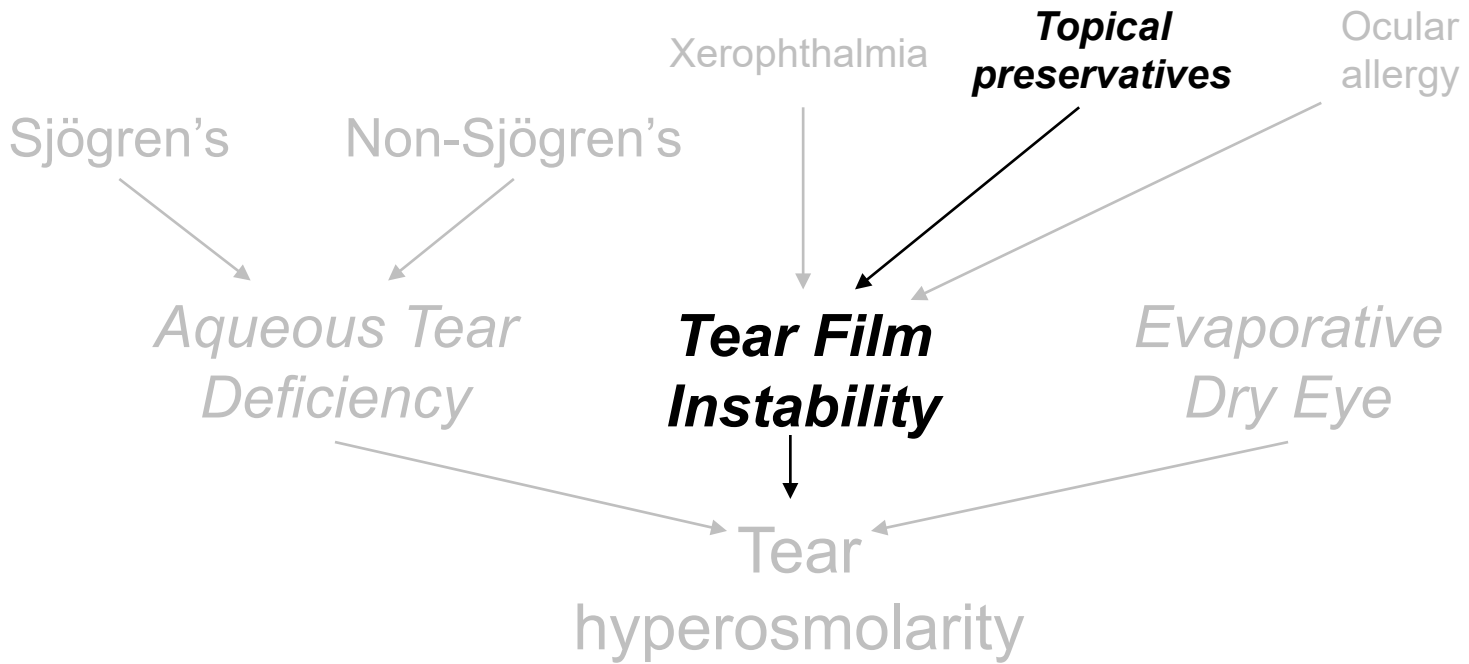
How do preservatives in ophthalmic preparations lead to TFI?



# Dry Eye Syndrome



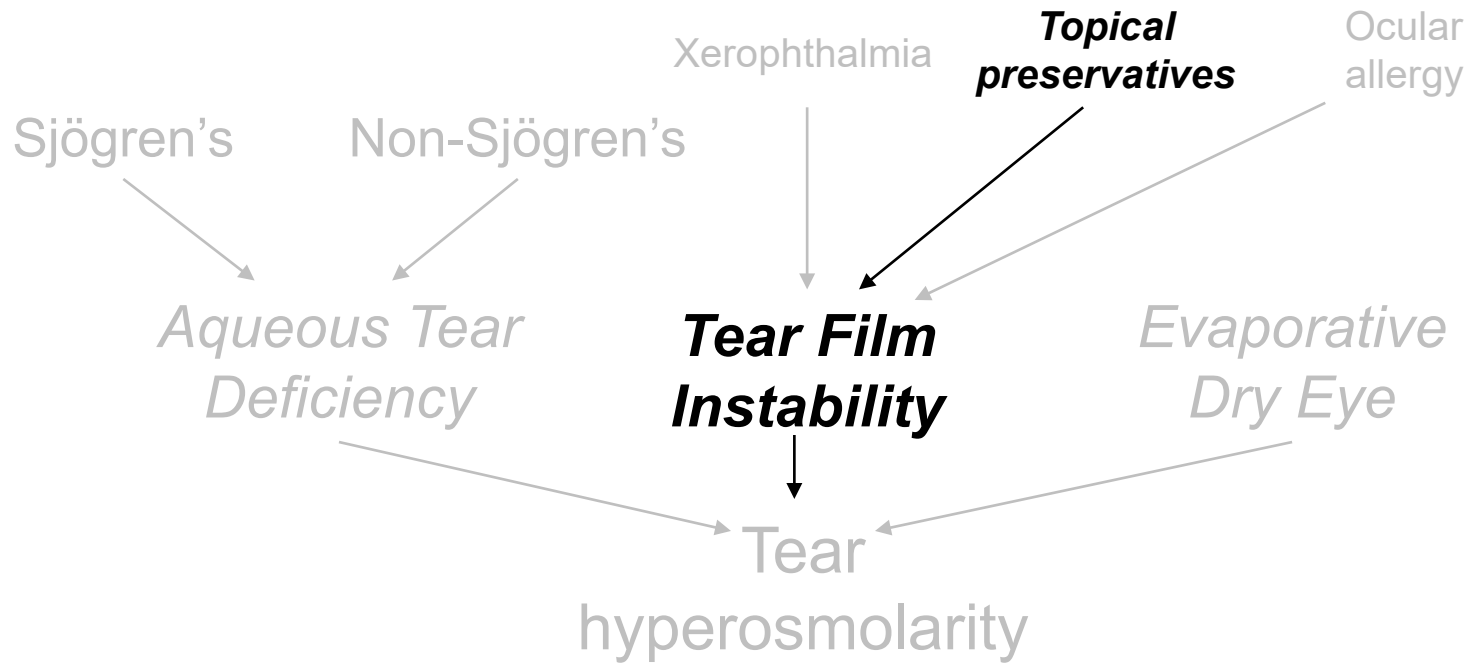
How do preservatives in ophthalmic preparations lead to TFI?  
By provoking an inflammatory response in the conj epithelium,  
which in turn promotes  cell apoptosis



# Dry Eye Syndrome



How do preservatives in ophthalmic preparations lead to TFI?  
By provoking an inflammatory response in the conj epithelium,  
which in turn promotes goblet cell apoptosis

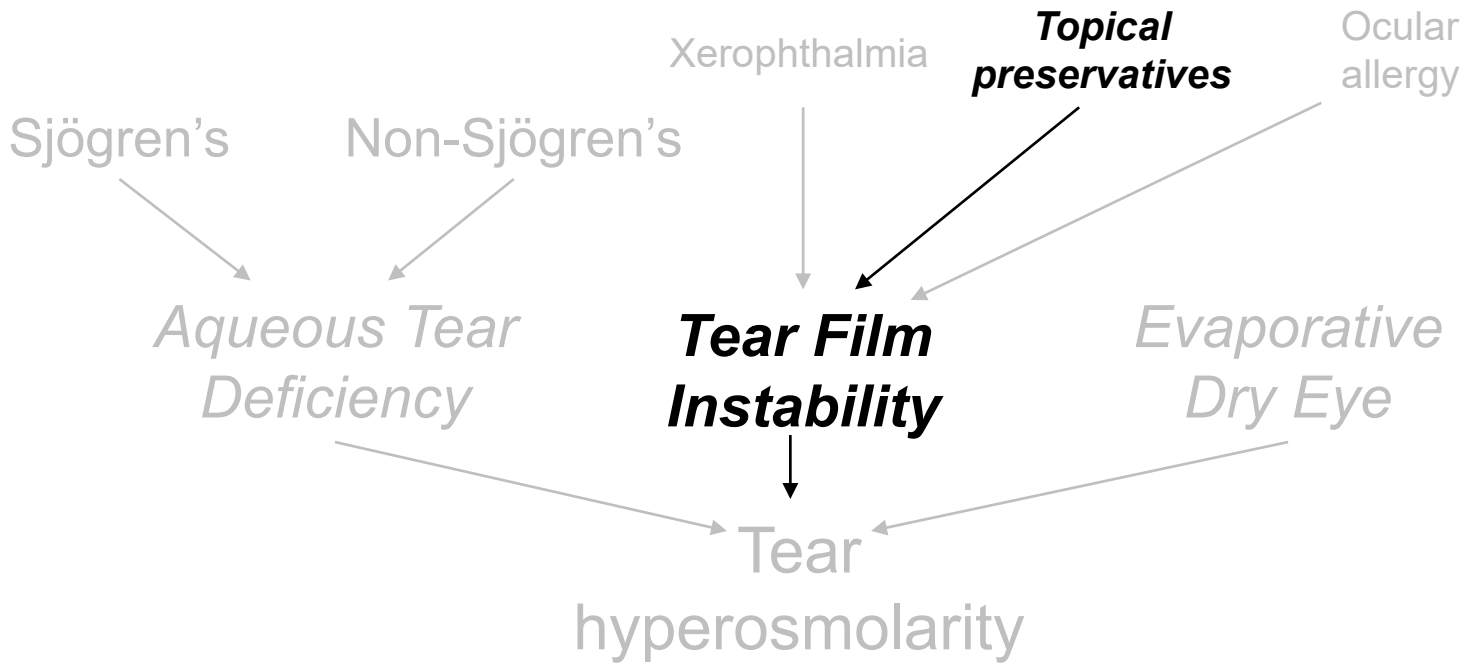


# Dry Eye Syndrome



*How do preservatives in ophthalmic preparations lead to TFI?*  
By provoking an inflammatory response in the conj epithelium,  
which in turn promotes goblet cell apoptosis

*Is there a preservative that is especially notorious for doing this?*

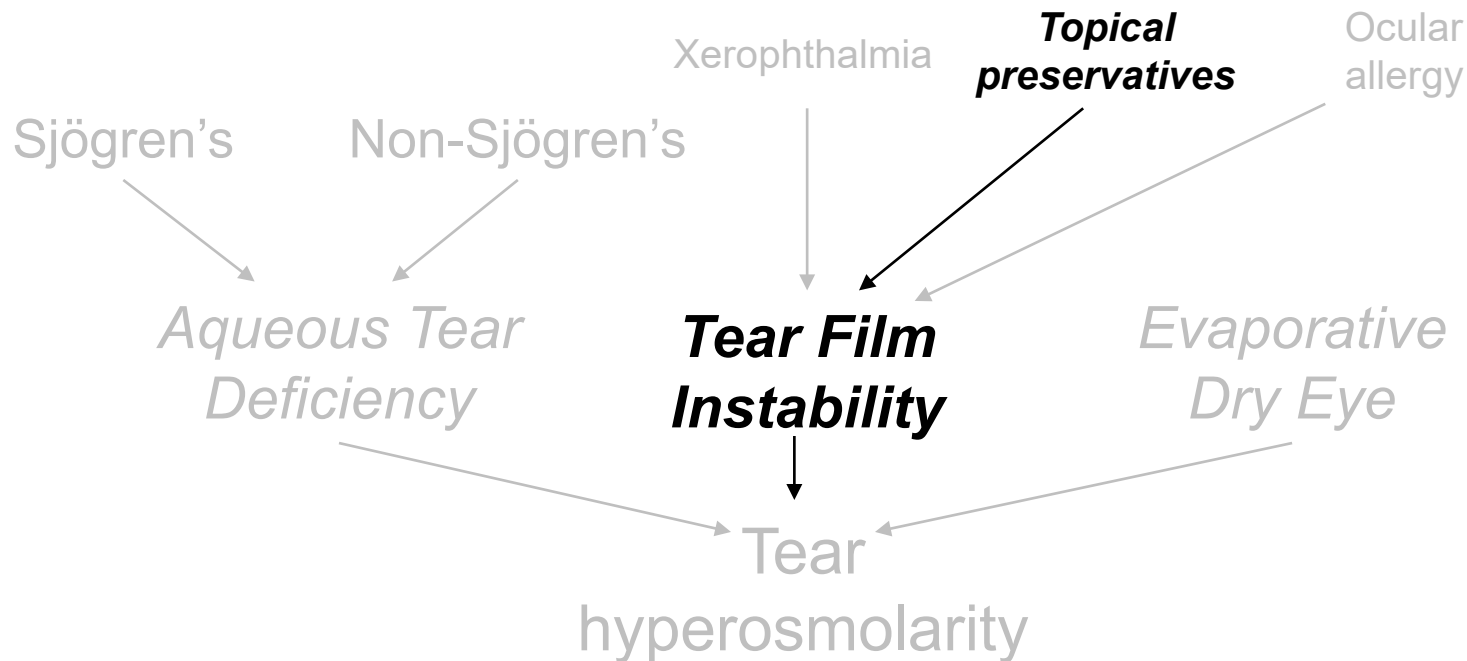


# Dry Eye Syndrome



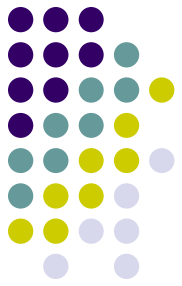
*How do preservatives in ophthalmic preparations lead to TFI?*  
By provoking an inflammatory response in the conj epithelium,  
which in turn promotes goblet cell apoptosis

*Is there a preservative that is especially notorious for doing this?*  
Benzalkonium chloride (aka BAK or BAC)

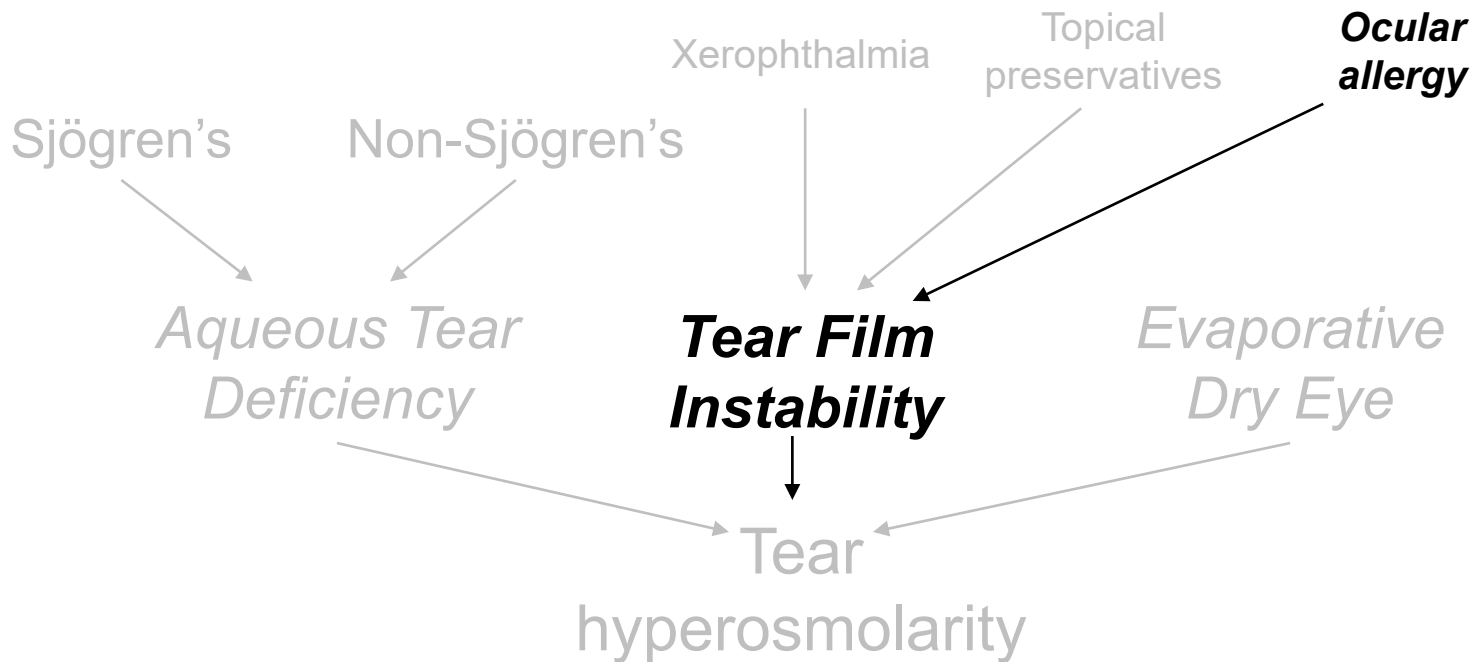




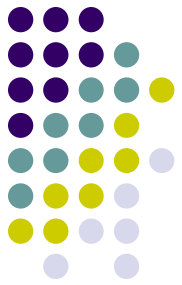
# Dry Eye Syndrome



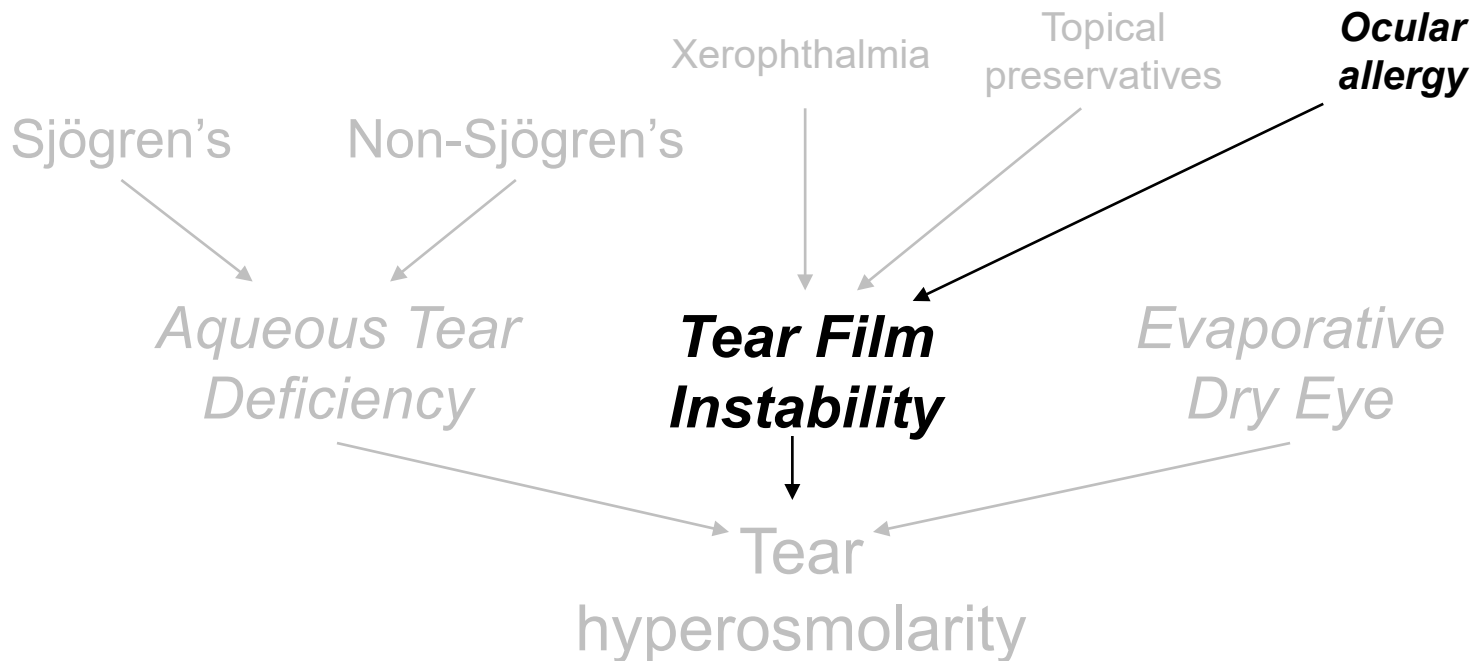
*How does an ocular allergic condition produce TFI?*



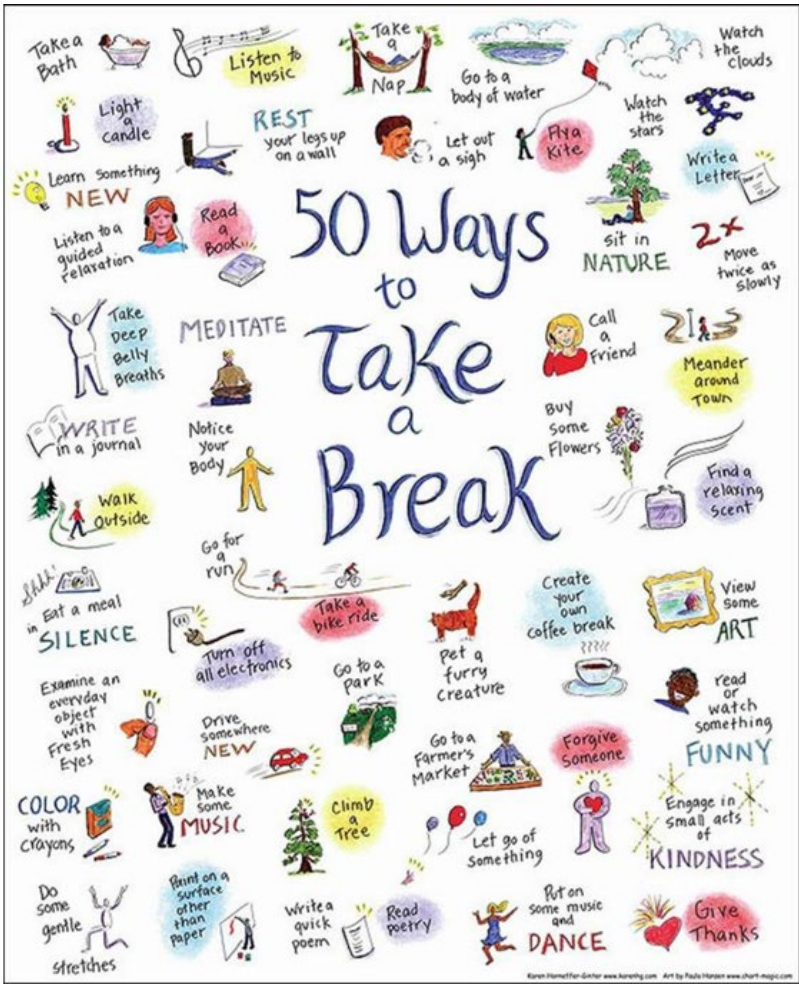
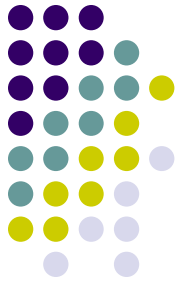
# Dry Eye Syndrome



*How does an ocular allergic condition produce TFI?*  
Allergen antigens on the ocular surface initiate an IgE-mediated inflammatory cascade, leading to goblet-cell loss

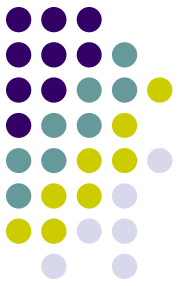


# Dry Eye Syndrome

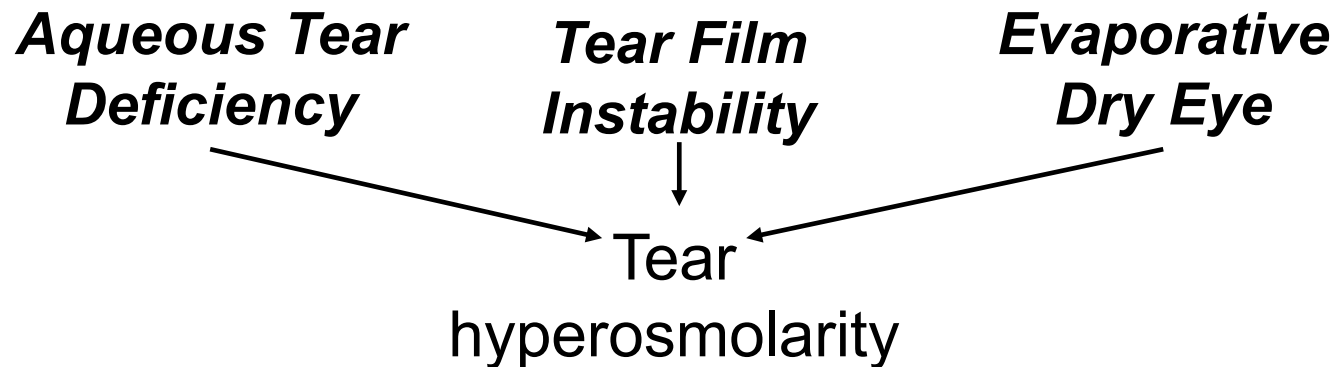


(This is a good point in the set to take a break)

# Dry Eye Syndrome



*Now that we understand how ATD, TFI and EDE lead to tear hyperosmolarity...*



# Dry Eye Syndrome



Now that we understand how ATD, TFI and EDE lead to tear hyperosmolarity...  
Let's examine how **tear hyperosmolarity** leads to **DES**

*Aqueous Tear  
Deficiency*

*Tear Film  
Instability*

*Evaporative  
Dry Eye*

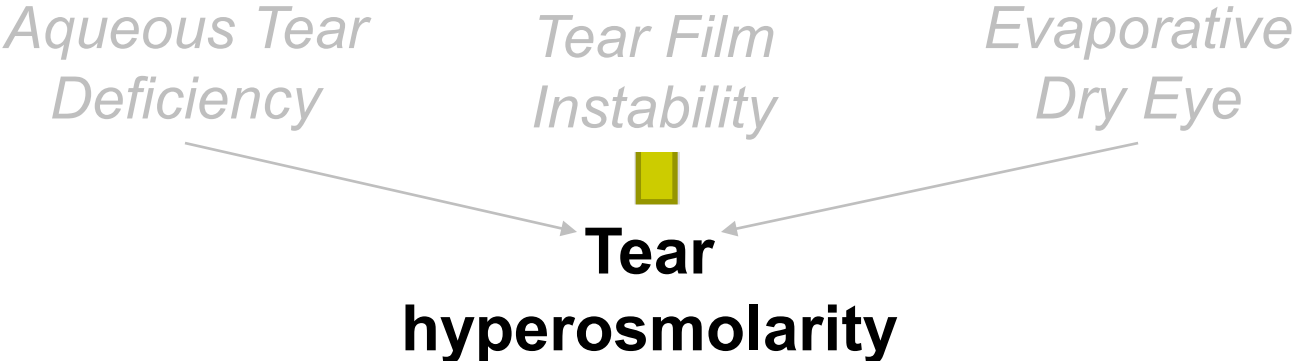
**Tear  
hyperosmolarity**



# Dry Eye Syndrome



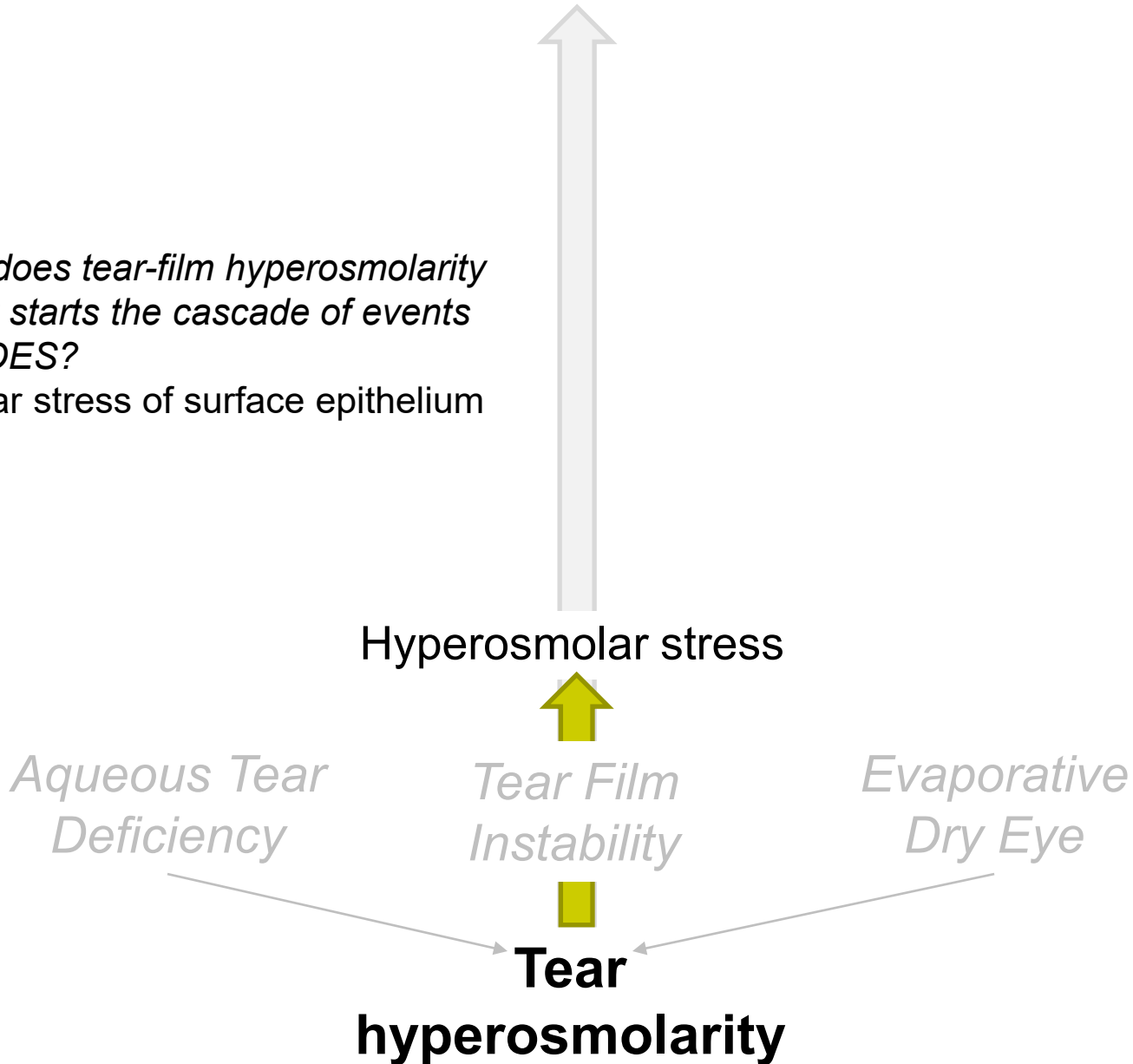
*What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?*



# Dry Eye Syndrome



*What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?*  
Hyperosmolar stress of surface epithelium

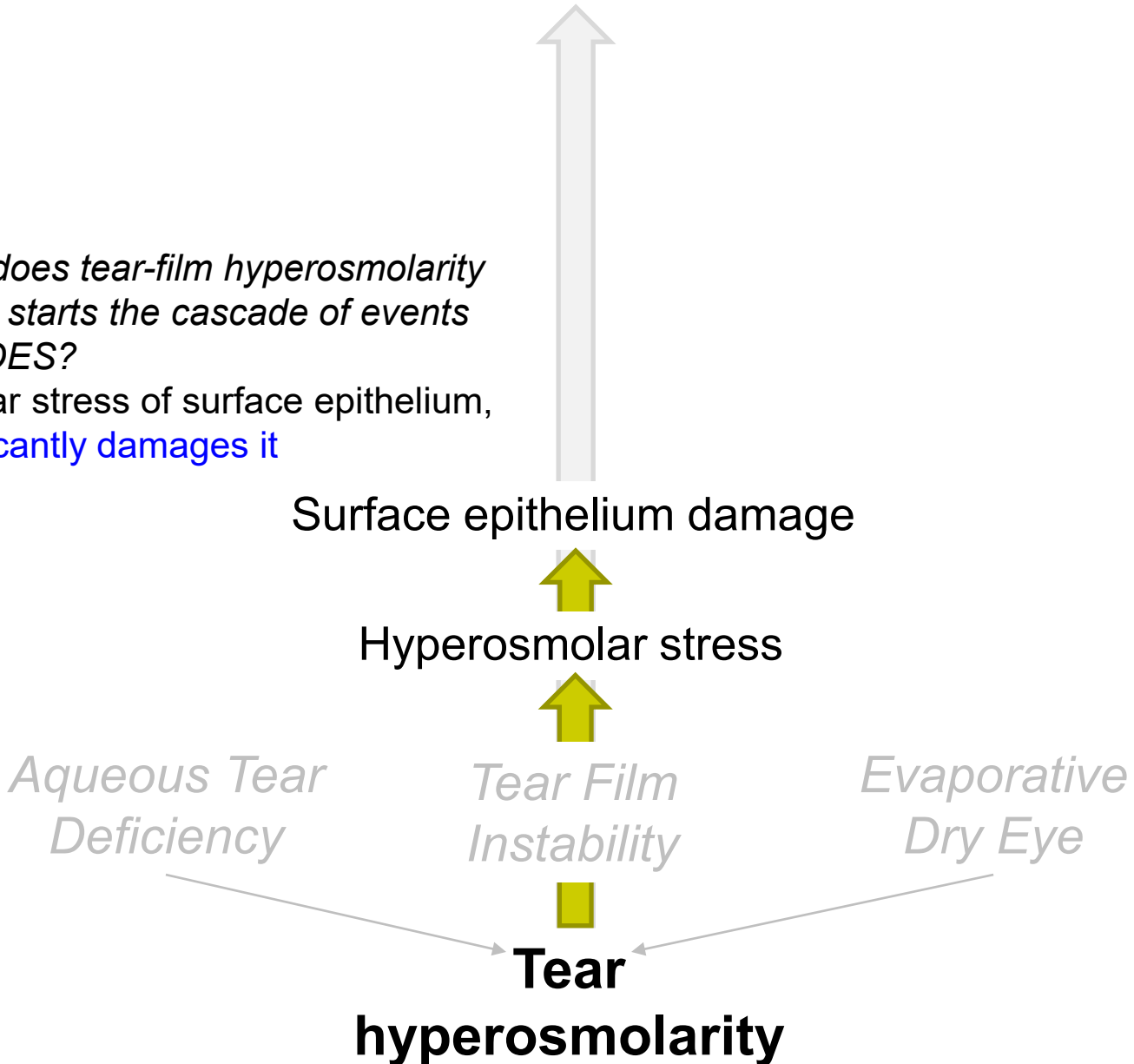


# Dry Eye Syndrome



*What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?*

Hyperosmolar stress of surface epithelium, which significantly damages it



Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

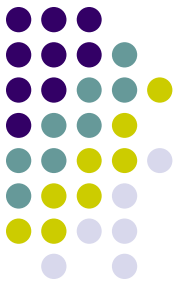
*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

*What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?*

**Hyperosmolar stress of surface epithelium,**

*which significantly damages it.*

*Are we talking corneal epi, or conj epi?*

Surface epithelium damage

Hyperosmolar stress

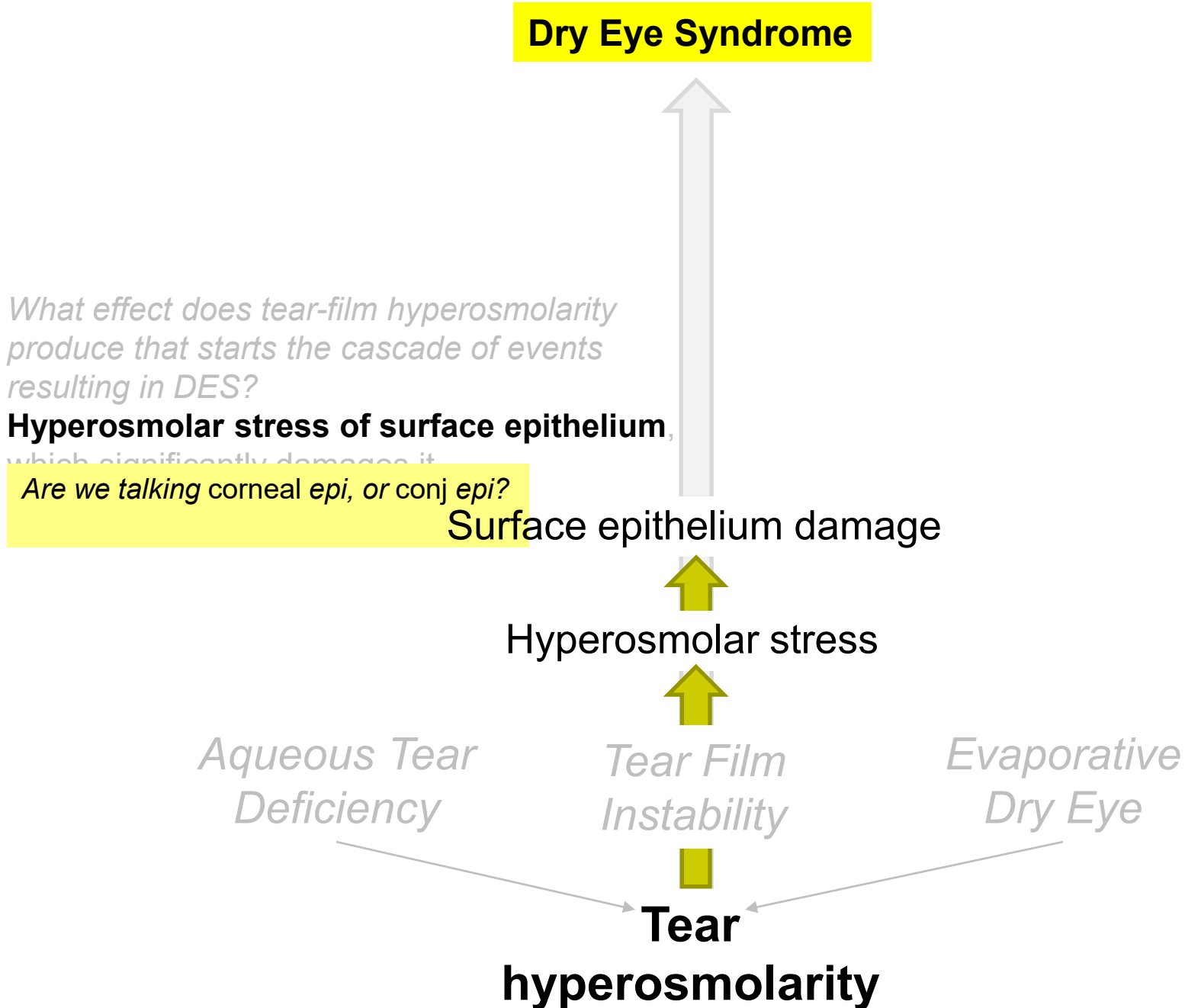
*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

*What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?*

**Hyperosmolar stress of surface epithelium,**  
*which significantly damages it*

*Are we talking corneal epi, or conj epi?*  
Both

Surface epithelium damage

Hyperosmolar stress

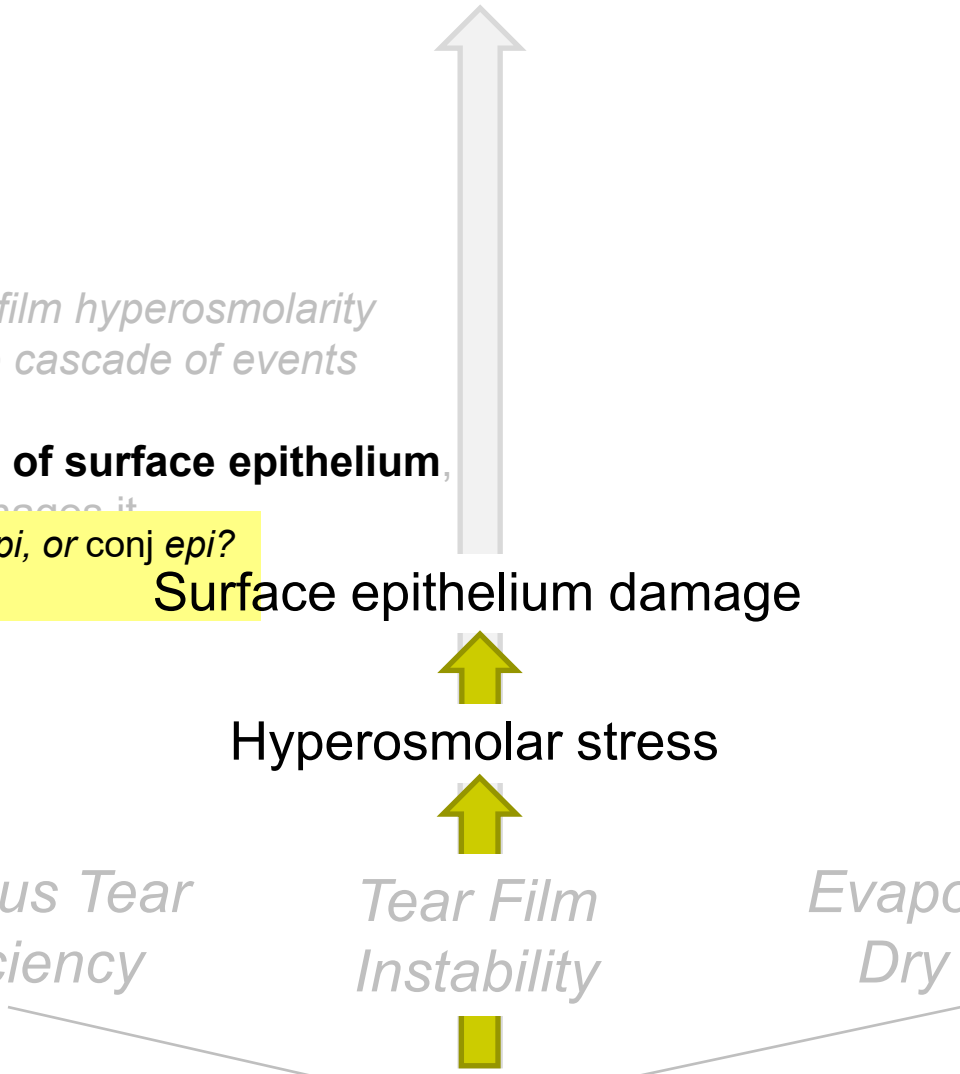
*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



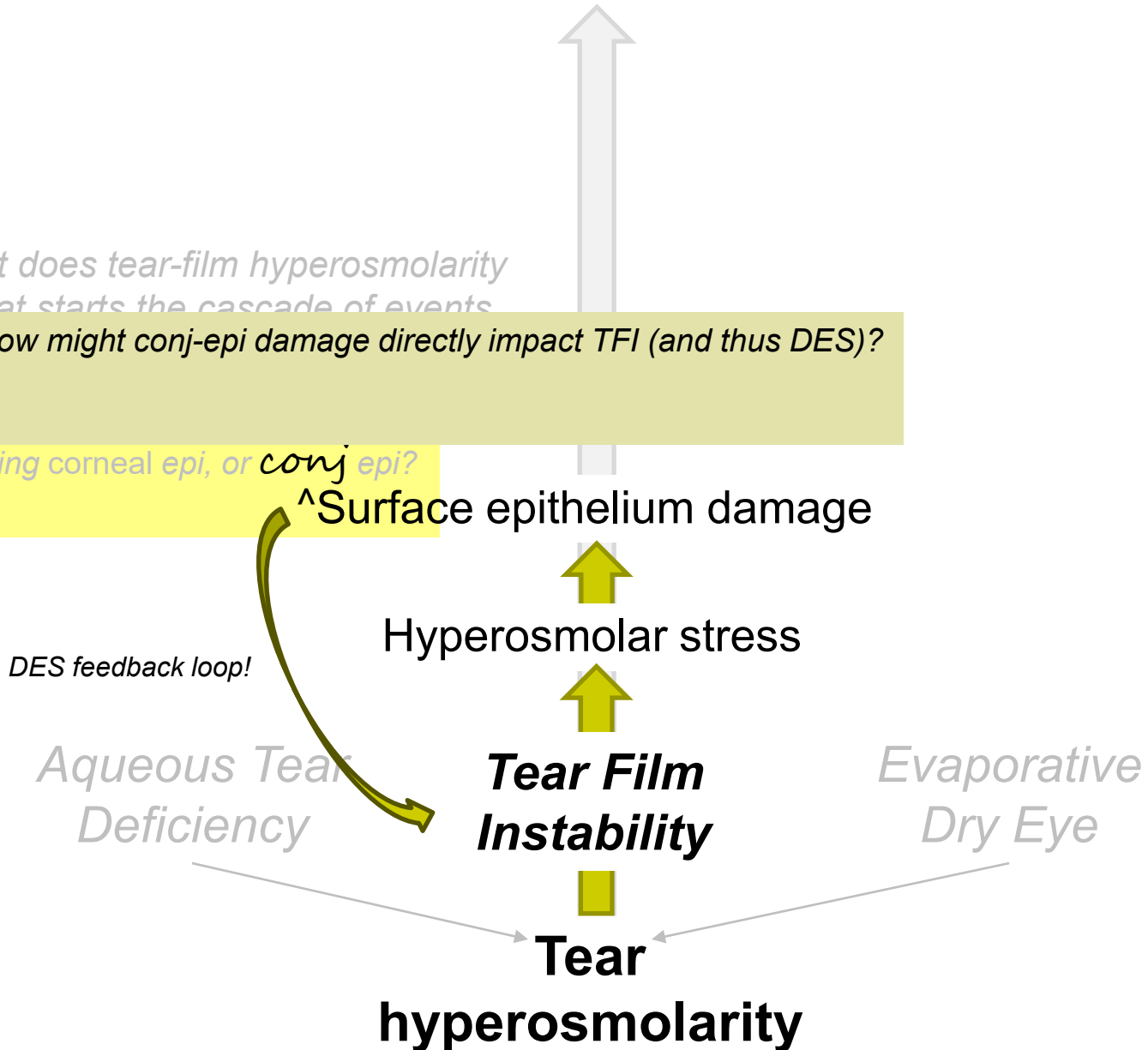


# Dry Eye Syndrome

What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?  
Hyperosmolarity

How might conj-epi damage directly impact TFI (and thus DES)?

Are we talking corneal epi, or conj epi?  
Both



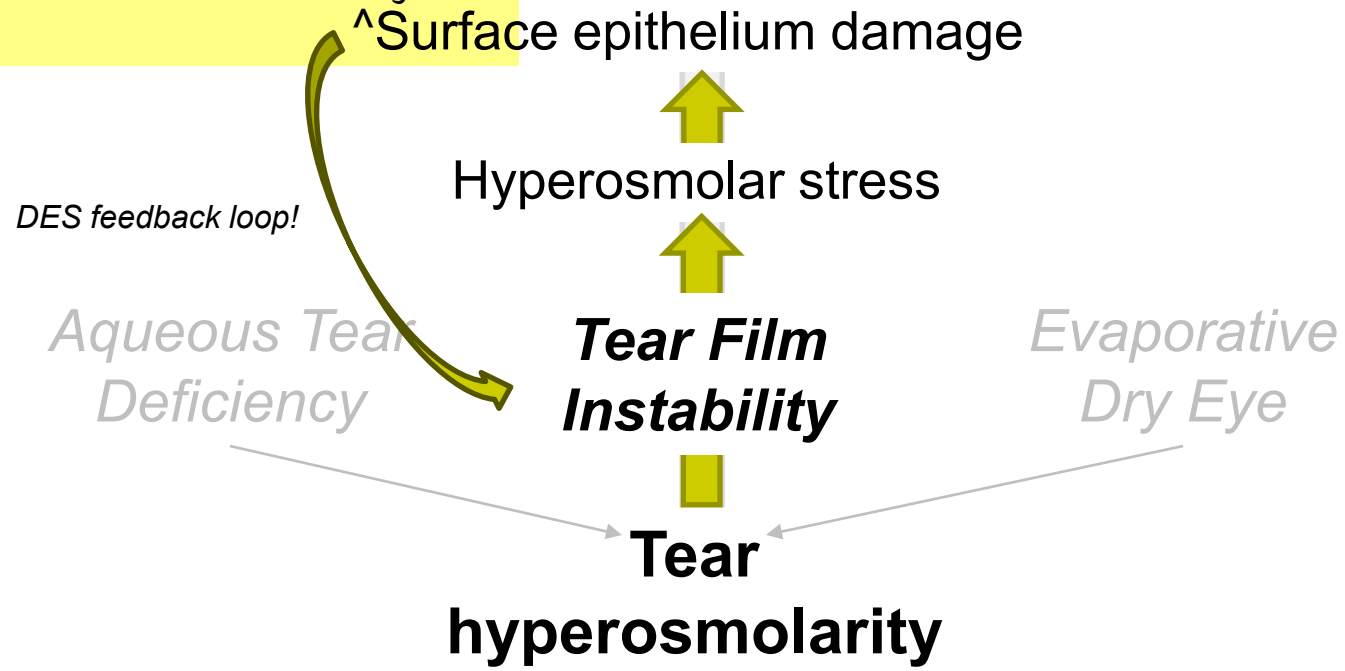


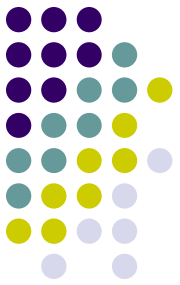
# Dry Eye Syndrome

What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?  
Hyperosmolarity

How might conj-epi damage directly impact TFI (and thus DES)?  
Recall that conj [ ] cells are the source of [ ], a deficit of which contributes to TFI and thus DES

Are we talking corneal epi, or conj epi?  
Both



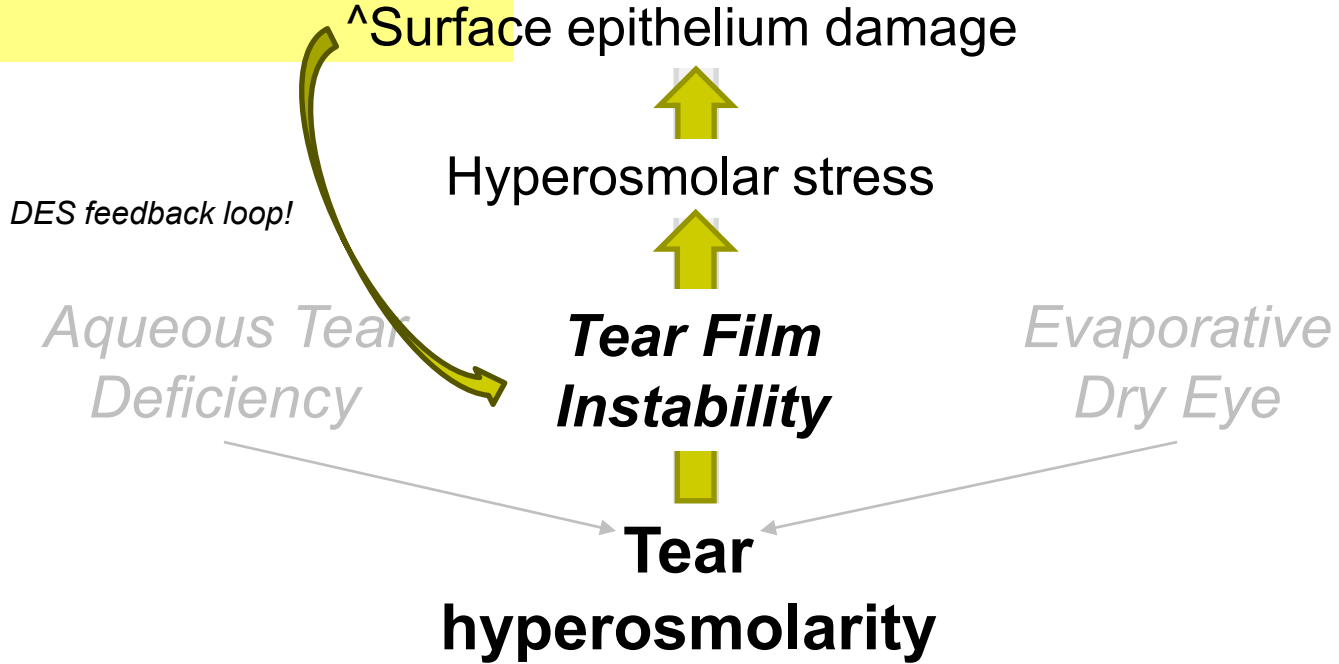


# Dry Eye Syndrome

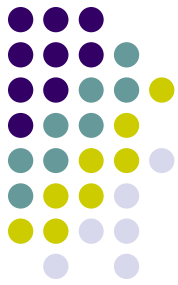
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?

How might conj-epi damage directly impact TFI (and thus DES)? Recall that conj goblet cells are the source of mucin, a deficit of which contributes to TFI and thus DES

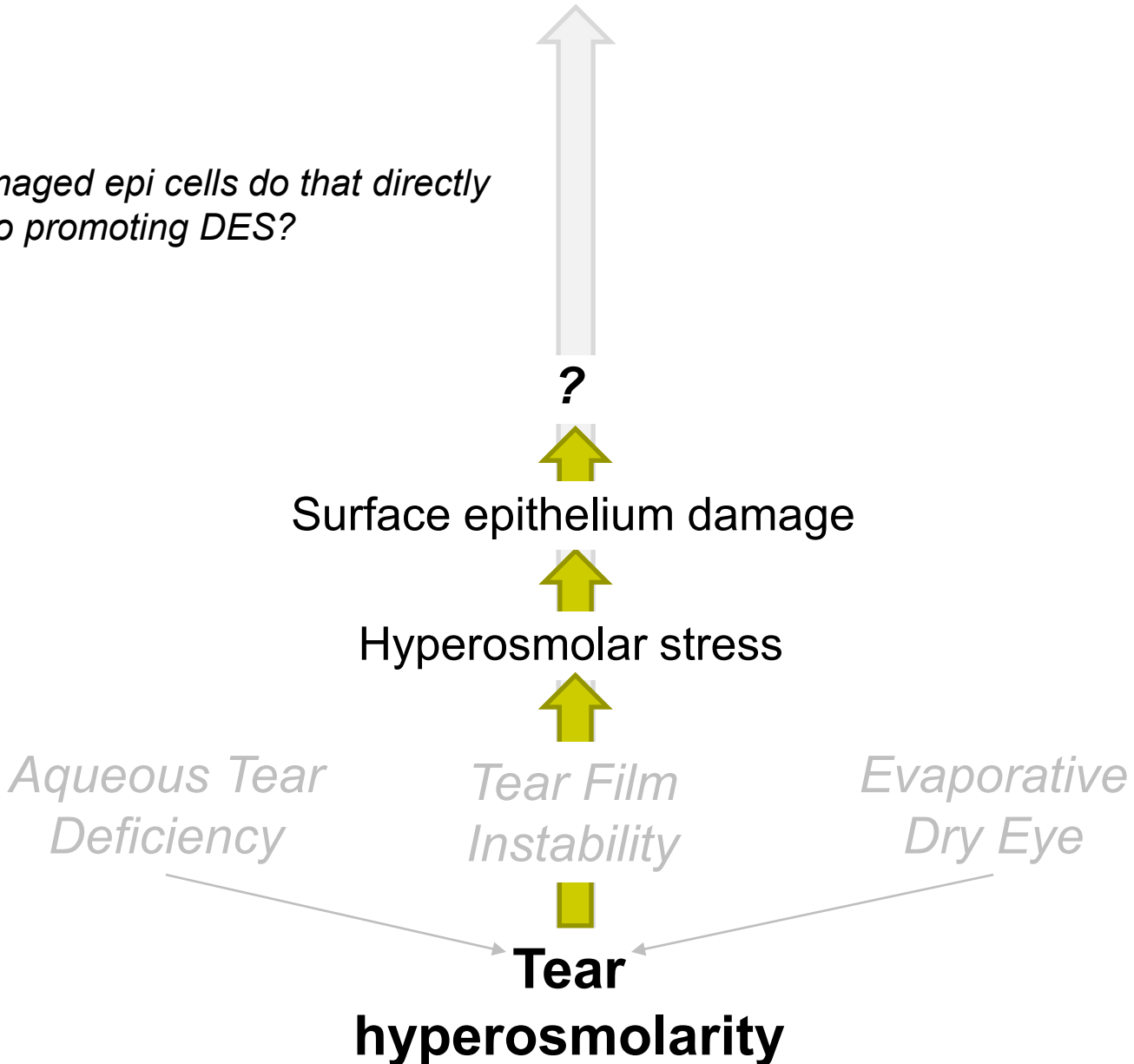
Are we talking corneal epi, or conj epi?  
Both



# Dry Eye Syndrome



*What do damaged epi cells do that directly contributes to promoting DES?*



# Dry Eye Syndrome



*What do damaged epi cells do that directly contributes to promoting DES?*

They release  that promote and/or facilitate inflammation

Inflammatory  release

Surface epithelium damage

Hyperosmolar stress

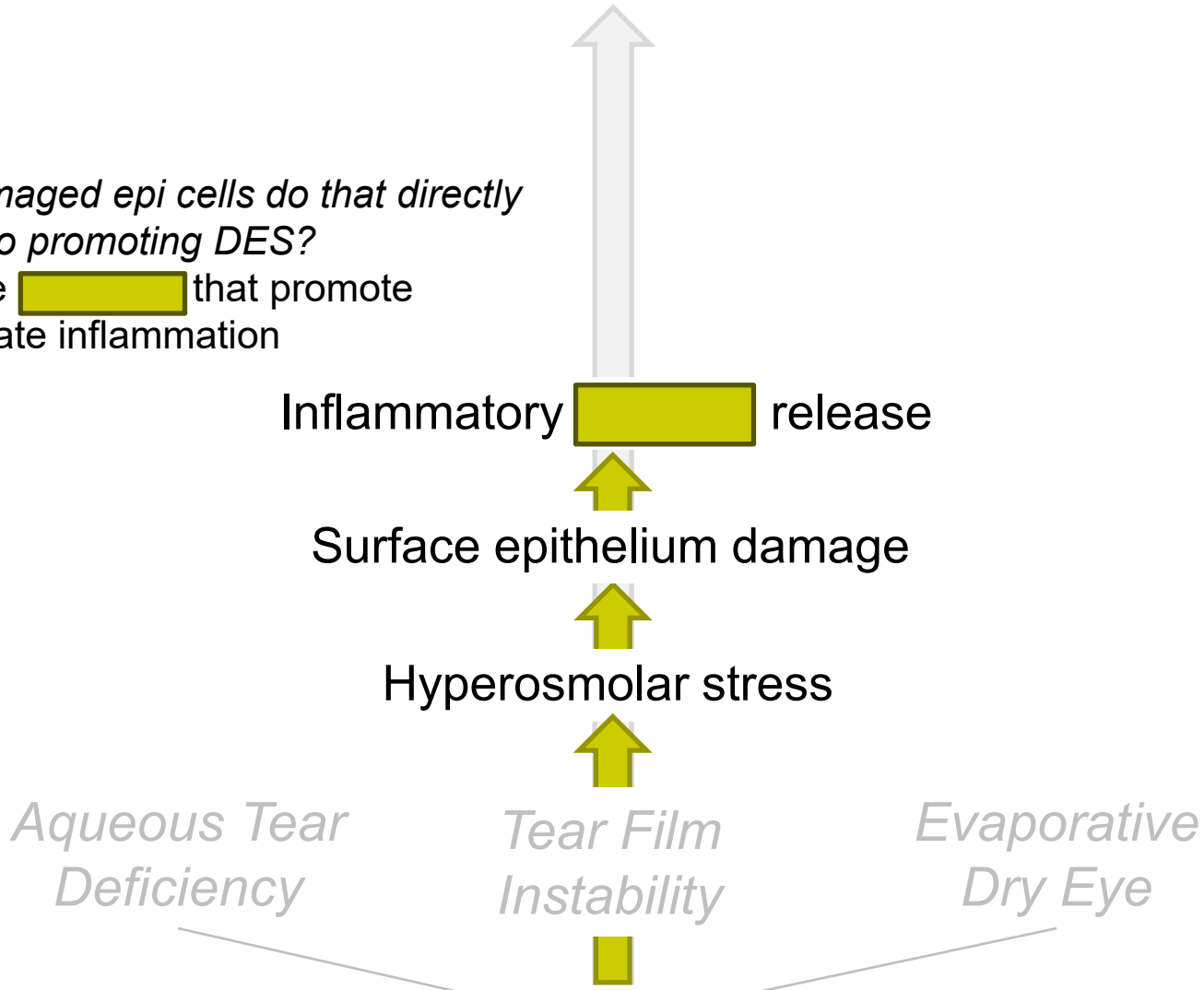
*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



*What do damaged epi cells do that directly contributes to promoting DES?*

They release cytokines that promote and/or facilitate inflammation



Inflammatory cytokine release



Surface epithelium damage



Hyperosmolar stress



*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*



**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

While a number of cytokines are released, the BCSC emphasizes # .

What do damaged epi cells contribute to promoting DE? They release cytokines that irritate and/or facilitate inflammation.

**cytokines**

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



*What do damaged epi cells contribute to promoting DE? They release cytokines that irritate and/or facilitate inflammation.*

*While a number of cytokines are released, the BCSC emphasizes three.*

**cytokines**

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

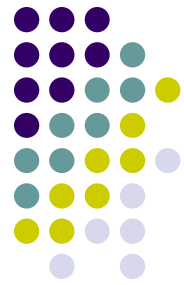
*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that irritate and/or facilitate inflammation.

**cytokines**

While a number of cytokines are released, the BCSC emphasizes three. *What are they?*  
--?  
--?  
--?

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

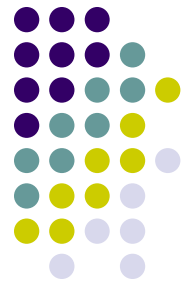
*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that and/or facilitate inflammation

**cytokines**

While a number of cytokines are released, the BCSC emphasizes three. *What are they?*  
--TNF  
--MMP-#  
--IL-#

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

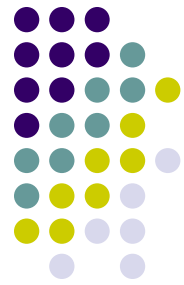
*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that irritate and/or facilitate inflammation.

**cytokines**

While a number of cytokines are released, the BCSC emphasizes three. *What are they?*  
--TNF  
--MMP-9  
--IL-1

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

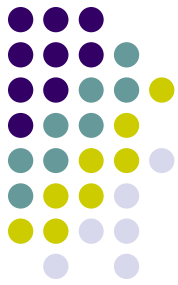
*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



What do damaged epi cells of the BCSC contribute to promoting DE? They release cytokines that can damage and/or facilitate inflammation.

While a number of cytokines are released, the BCSCs release --TNF, --MMP-9, and --IL-1.

What negative effects does MMP-9 have on the ocular surface?

Inflammation

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

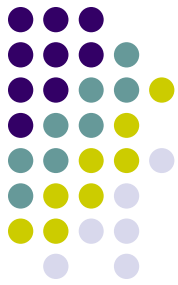
Evaporative Dry Eye

Tear

hyperosmolarity



# Dry Eye Syndrome



What do damaged epi cells of the BCSC contribute to promoting DE? They release cytokines that damage and/or facilitate inflammation

While a number of cytokines are released, the BCSCs release --TNF --MMP-9 --IL-1

What negative effects does MMP-9 have on the ocular surface? It cleaves epi cells from their BM, and from one another, by disrupting junctional elements

Inflammation

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

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hyperosmolarity



# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that and/or facilitate inflammation

**cytokines**

While a number of cytokines are released, the BCSC releases --TNF --MMP-9 --IL-1

What negative effects does MMP-9 have on the ocular surface? It cleaves epi cells from their BM, and from one another, by disrupting junctional elements

How do these effects manifest clinically, ie, at the slit lamp?

Inflammation

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

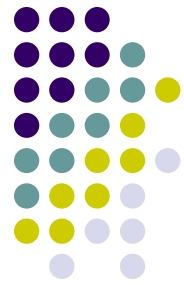
**Tear**

**hyperosmolarity**





# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that and/or facilitate inflammation

**cytokines**

While a number of cytokines are released, the BCSC --TNF --MMP-9 --IL-1

What negative effects does MMP-9 have on the ocular surface? It cleaves epi cells from their BM, and from one another, by disrupting junctional elements

How do these effects manifest clinically, ie, at the slit lamp? As increased fluorescein staining in the form of punctate epithelial erosions

Inflammation

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

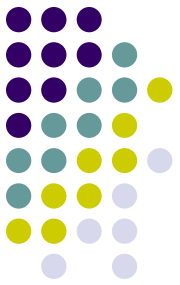
Tear Film Instability

Evaporative Dry Eye

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

While a number of cytokines are released,

*TNF and IL-1 have a variety of effects, but the BCSC dwells on one in particular—which is it?*

- TNF
- MMP
- IL-1

*What do damaged epi cells of the BCSC contribute to promoting DE? They release cytokines that and/or facilitate inflammation*

**cytokines**

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

While a number of cytokines are released,

TNF and IL-1 have a variety of effects, but the BCSC dwells on one in particular—which is it?  
Promotion of [redacted] among surface epi cells (which also leads to PEE)

What do damaged epi cells of the BCSC contribute to promoting DE? They release cytokines that and/or facilitate inflammation

**cytokines**

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

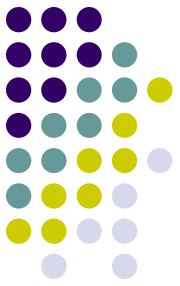
Tear Film Instability

Evaporative Dry Eye

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

While a number of cytokines are released,

*TNF and IL-1 have a variety of effects, but the BCSC dwells on one in particular—which is it?*

- TNF--
- MMP--
- IL-1--

Promotion of apoptosis among surface epi cells (which also leads to PEE)

*What do damaged epi cells of the BCSC contribute to promoting DE? They release cytokines that and/or facilitate inflammation*

**cytokines**

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**



# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE?  
They release **cytokines** that and/or facilitate inflammation

While a number of cytokines are released, the BCSC emphasizes three. What are they?  
--TNF  
--MMP-9  
--IL-1

Inflammatory cytokine release

Surface epithelium damage

Note that surface epi damage induces cytokine release...

*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that and/or facilitate inflammation

While a number of cytokines are released, the BCSC emphasizes three. What are they?  
--**TNF: Promotes apoptosis**  
--**MMP-9: Cleaves epi cells**  
--**IL-1: Promotes apoptosis**

Inflammatory cytokine release

Surface epithelium damage



DES feedback loop!

Note that surface epi damage induces cytokine release... *And cytokine release induces surface epi damage.*

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear

hyperosmolarity



# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that and/or facilitate inflammation

While a number of cytokines are released, the BCSC emphasizes three. What are they?  
--**TNF: Promotes apoptosis**  
--**MMP-9: Cleaves epi cells**  
--**IL-1: Promotes apoptosis**

Inflammatory cytokine release

Surface epithelium damage



DES feedback loop!

Note that surface epi damage induces cytokine release... **And cytokine release induces surface epi damage.** Thus, a vicious cycle/circle develops in which epi damage leads directly to further epi damage.

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear

hyperosmolarity

# Dry Eye Syndrome



What do damaged epi cells contribute to promoting DE? They release cytokines that and/or facilitate inflammation

While a number of cytokines are released, the BCSC emphasizes three. What are they?  
--**TNF: Promotes apoptosis**  
--**MMP-9: Cleaves epi cells**  
--**IL-1: Promotes apoptosis** } as well as...

Inflammatory cytokine release

**Note: Cytokines play another role in DES pathogenesis, one so important that we're going to discuss it separately. Stay tuned.**

Note that surface epi damage induces cytokine release... And cytokine release induces surface epi damage. Thus, a vicious cycle/circle develops in which epi damage leads directly to further epi damage.

Aqueous Tear Deficiency

Tear Film Instability

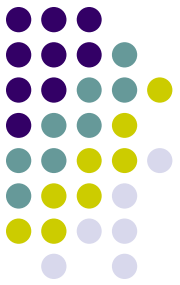
Evaporative Dry Eye

Tear

hyperosmolarity



# Dry Eye Syndrome



*In addition to cytokine production, surface-epi damage promotes the expression of a particular 'adhesion' molecule—which one?*

Inflammatory *adhesion molecule?*

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear  
Deficiency*

*Tear Film  
Instability*

*Evaporative  
Dry Eye*

**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



*In addition to cytokine production, surface-epi damage promotes the expression of a particular 'adhesion' molecule—which one?*  
Intercellular adhesion molecule 1 (ICAM-1)

Inflammatory adhesion molecule ICAM-1

Surface epithelium damage

Hyperosmolar stress

*Aqueous Tear  
Deficiency*

*Tear Film  
Instability*

*Evaporative  
Dry Eye*

**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



*In addition to cytokine production, surface-epi damage promotes the expression of a particular 'adhesion' molecule—which one? Intercellular adhesion molecule 1 (ICAM-1)*

## Inflammatory adhesion molecule *ICAM-1*

*Increased ICAM-1 expression on two cell types are of particular importance vis a vis DES—which cell types?*

*Aqueous Tear Deficiency*

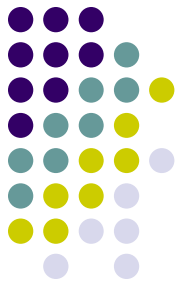
*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



*In addition to cytokine production, surface-epi damage promotes the expression of a particular 'adhesion' molecule—which one? Intercellular adhesion molecule 1 (ICAM-1)*

## Inflammatory adhesion molecule *ICAM-1*

*Increased ICAM-1 expression on two cell types are of particular importance vis a vis DES—which cell types? Vascular endothelial cells and T-lymphocytes*

*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*



**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



*In addition to cytokine production, surface-epi damage promotes the expression of a particular 'adhesion' molecule—which one? Intercellular adhesion molecule 1 (ICAM-1)*

## Inflammatory adhesion molecule *ICAM-1*

*Increased ICAM-1 expression on two cell types are of particular importance vis a vis DES—which cell types?*

Vascular endothelial cells and T-lymphocytes

*Why is ICAM-1 expression on these cells particularly important in the pathophysiology of DES?*

*Aqueous Tear  
Deficiency*

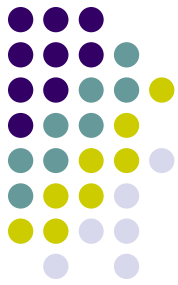
*Tear Film  
Instability*

*Evaporative  
Dry Eye*

**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



*In addition to cytokine production, surface-epi damage promotes the expression of a particular 'adhesion' molecule—which one? Intercellular adhesion molecule 1 (ICAM-1)*

## Inflammatory adhesion molecule *ICAM-1*

*Increased ICAM-1 expression on two cell types are of particular importance vis a vis DES—which cell types?  
Vascular endothelial cells and T-lymphocytes*

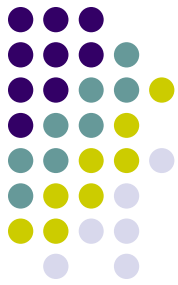
*Why is ICAM-1 expression on these cells particularly important in the pathophysiology of DES?  
Because it promotes/facilitates T-cell migration to the ocular surface and lacrimal glands, where they play a central role in the inflammatory response*

*Aqueous tear Deficiency      Tear Film Instability      Evaporative Dry Eye*

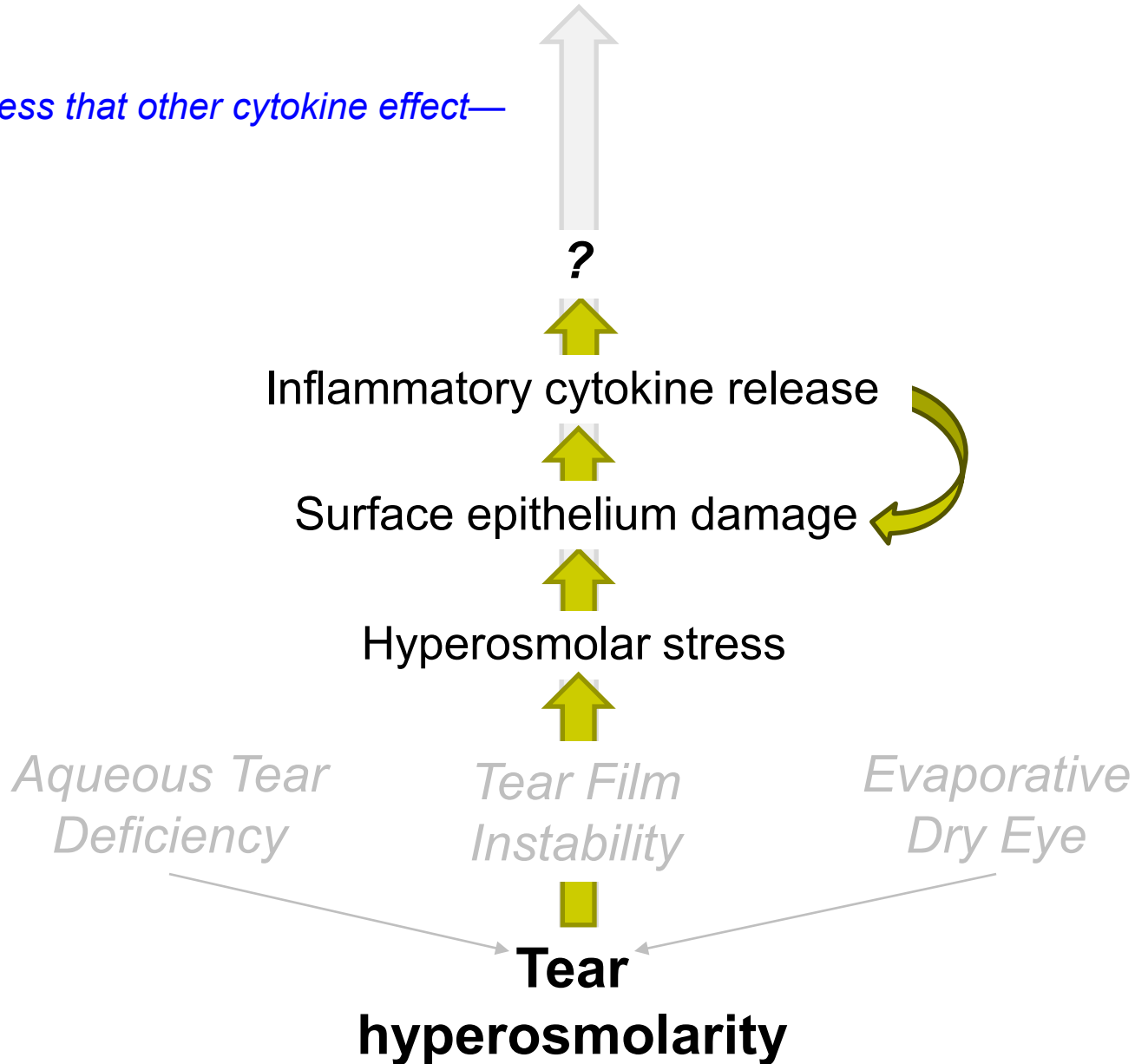


**Tear hyperosmolarity**

# Dry Eye Syndrome



*Now to address that other cytokine effect—  
what is it?*

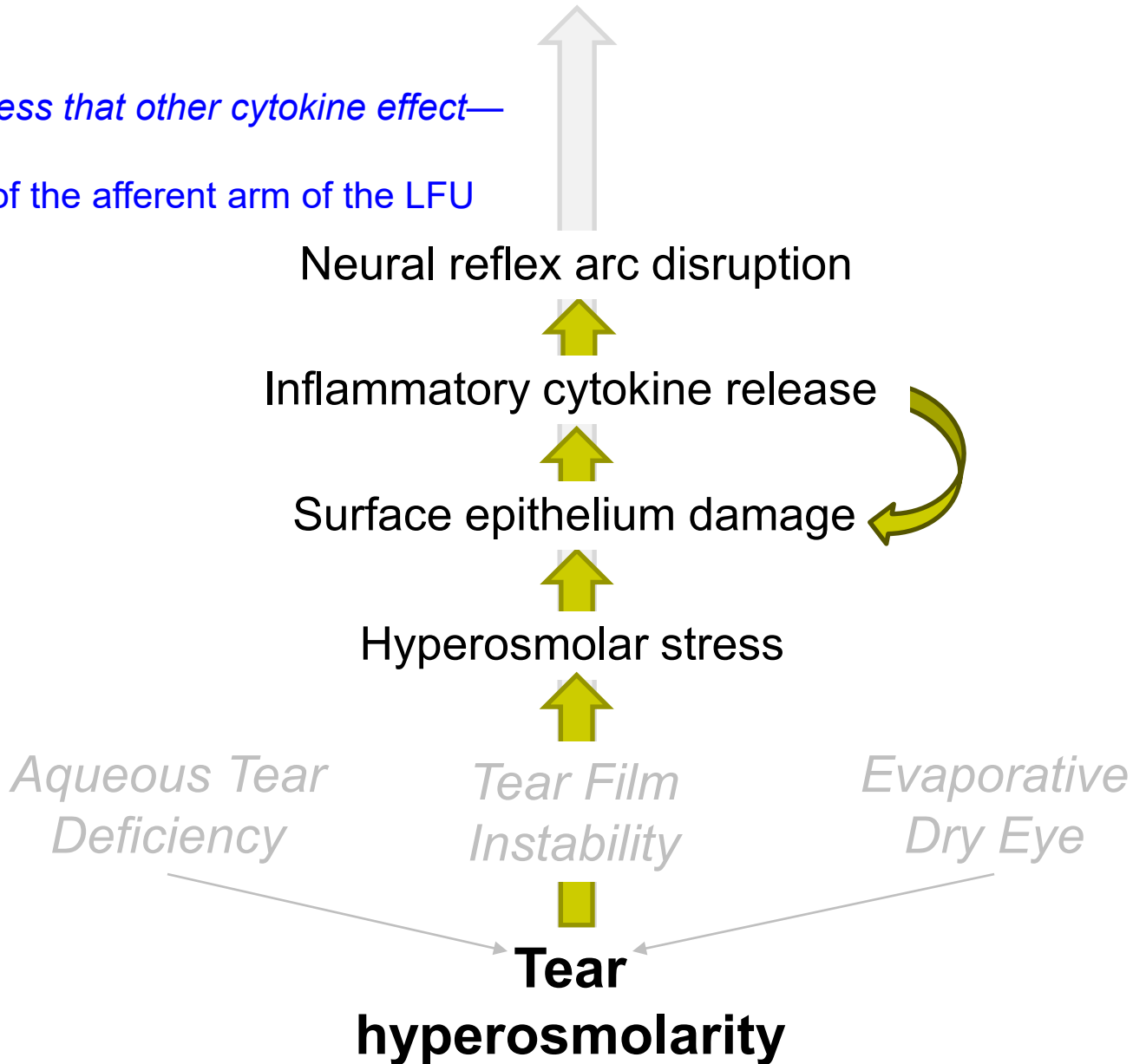


# Dry Eye Syndrome



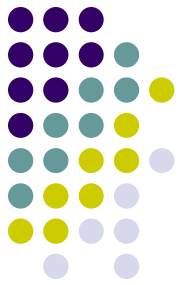
*Now to address that other cytokine effect—  
what is it?*

Impedance of the afferent arm of the LFU  
reflex arc





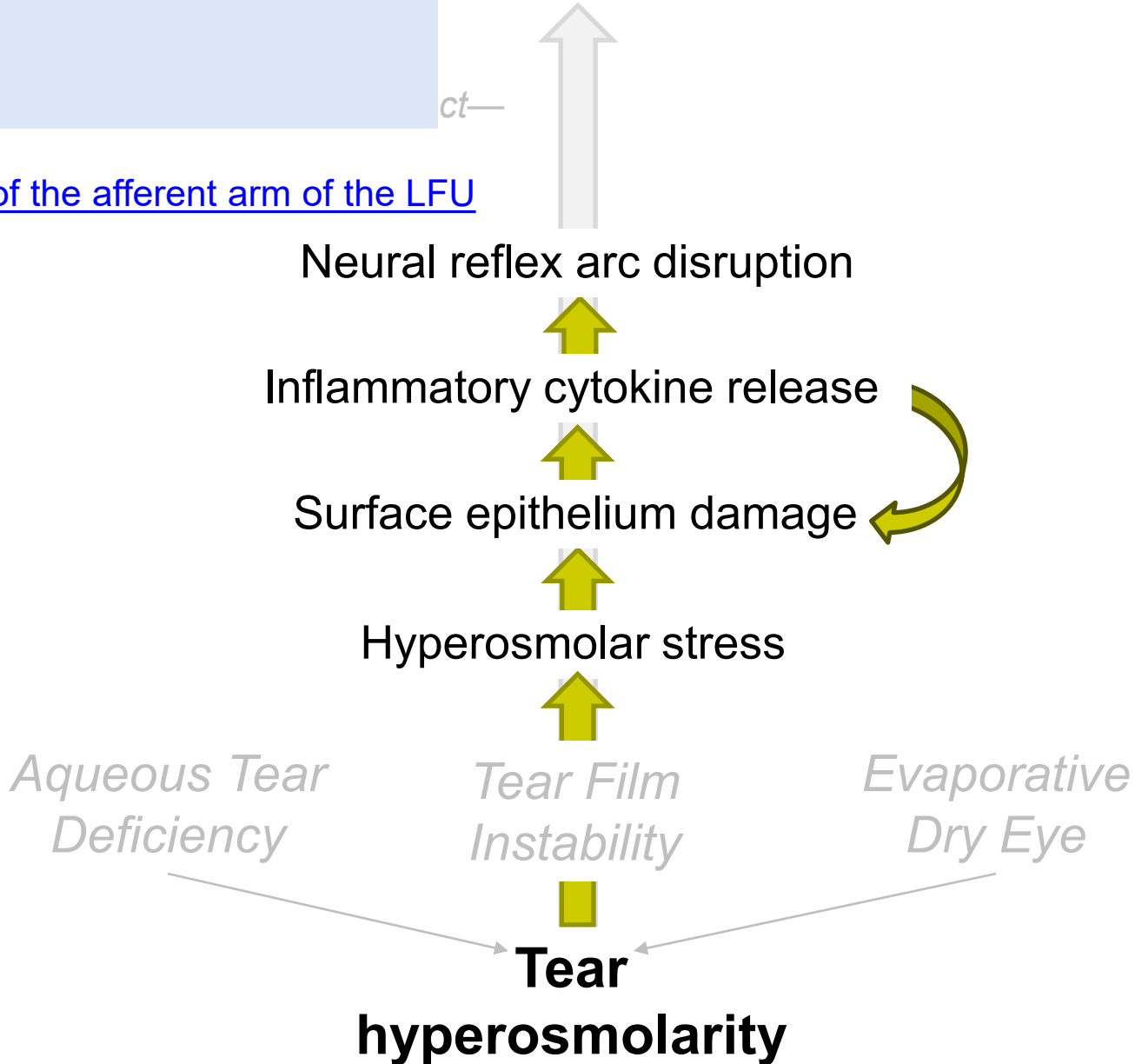
# Dry Eye Syndrome



How does this come about?

what is it?

Impedance of the afferent arm of the LFU reflex arc

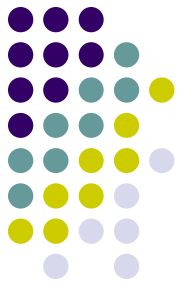
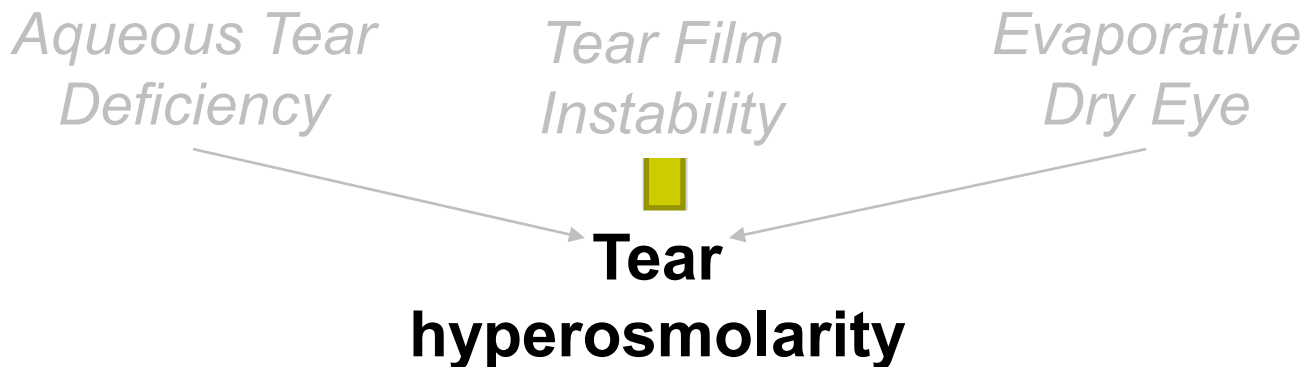


# Dry Eye Syndrome

How does this come about?  
The BCSC is vague on this score, stating simply that 'inflammatory cytokines block neural signals for tear secretion'

what is it?

Impedance of the afferent arm of the LFU reflex arc



*Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?*

## Dry Eye Syndrome



Neural reflex arc disruption



Inflammatory cytokine release



Surface epithelium damage



Hyperosmolar stress



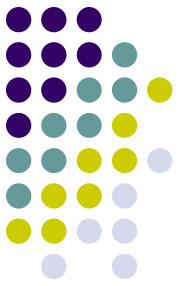
*Aqueous Tear Deficiency*

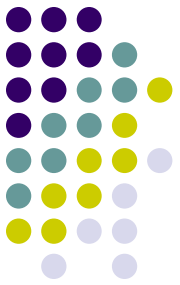
*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

*Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?*

*Decrease in aqueous production by the lac glands*

Decreased aqueous production

Neural reflex arc disruption

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

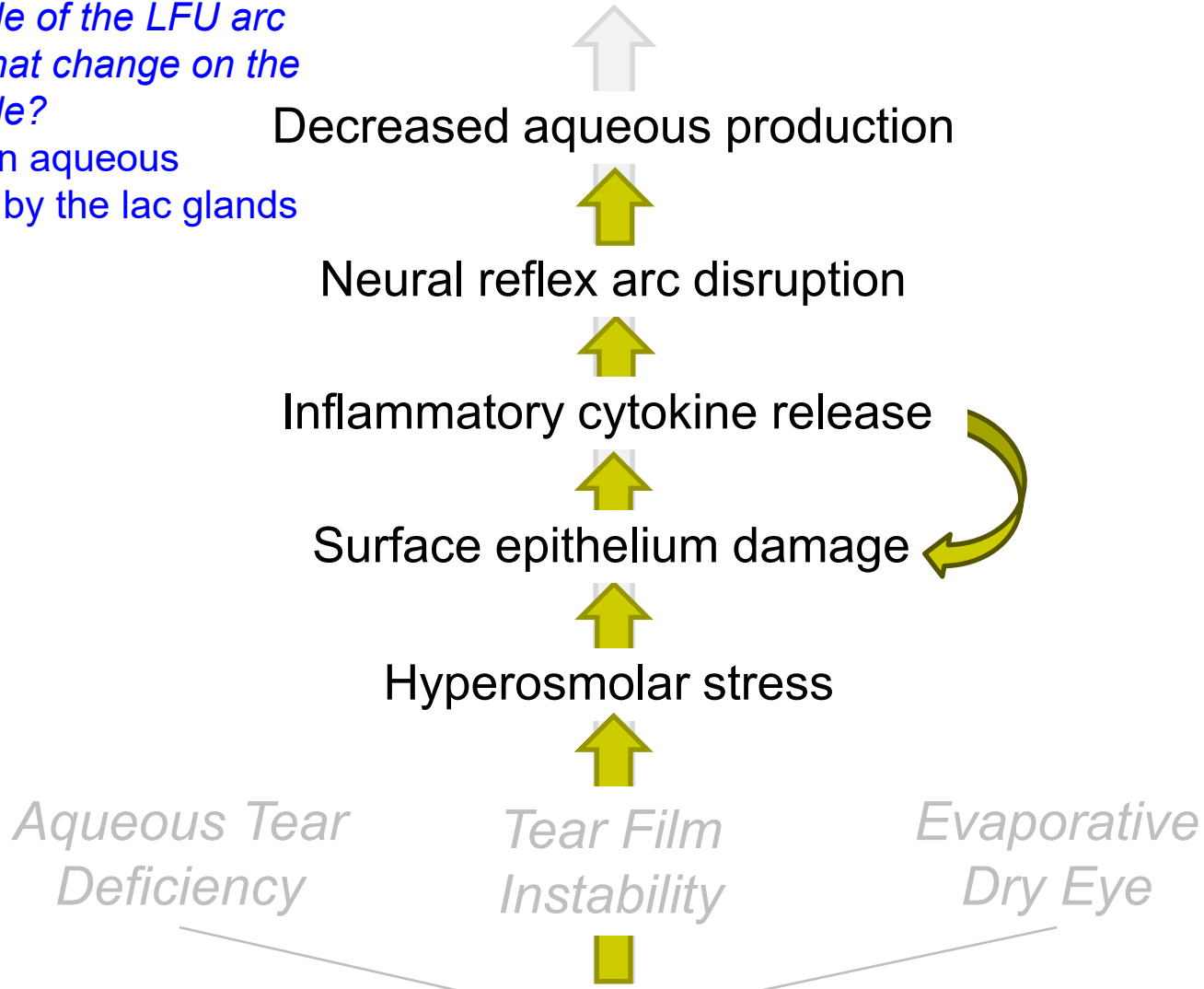
*Aqueous Tear Deficiency*

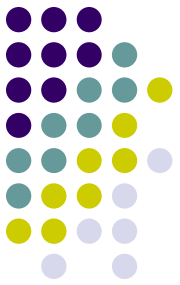
*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**





# Dry Eye Syndrome

*Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?*

**Decrease in aqueous production by the lac glands**

Decreased aqueous production



*Hold up—if aqueous production is suppressed, how come so many DES pts present with excessive tearing?*



Hyperosmolar stress



*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*



**Tear**

**hyperosmolarity**



# Dry Eye Syndrome

*Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?*

**Decrease in aqueous production by the lac glands**

Decreased aqueous production



*Hold up—if aqueous production is suppressed, how come so many DES pts present with excessive tearing?*  
Early in the DES course there is an inflammation-driven uptick in corneal-nerve activity that increases reflex-driven lacrimal gland stimulation, which produces the oft-observed DES pt c/o of tearing (tl;dr irritated eyes often run water).



Hyperosmolar stress



*Aqueous Tear Deficiency*

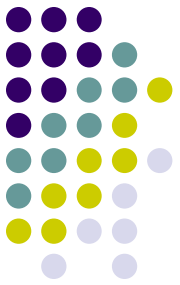
*Tear Film Instability*

*Evaporative Dry Eye*



**Tear**

**hyperosmolarity**



# Dry Eye Syndrome

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Decreased aqueous production

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Early in the DES course there is an inflammation-driven uptick in corneal-nerve activity that increases reflex-driven lacrimal gland stimulation, which produces the oft-observed DES pt c/o of tearing (tl;dr irritated eyes often run water).

**Later** in the dz process, cumulative nerve damage leads to a diminution in afferent input and thus a **decrease** in lac gland stimulation, resulting in the decrease in aqueous production as described here.

Hyperosmolar stress

*Aqueous Tear Deficiency*

*Tear Film Instability*

*Evaporative Dry Eye*

**Tear**

**hyperosmolarity**

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



Note that neural reflex arc disruption decreases aqueous production...



*Aqueous Tear  
Deficiency*

*Tear Film  
Instability*

*Evaporative  
Dry Eye*

**Tear**

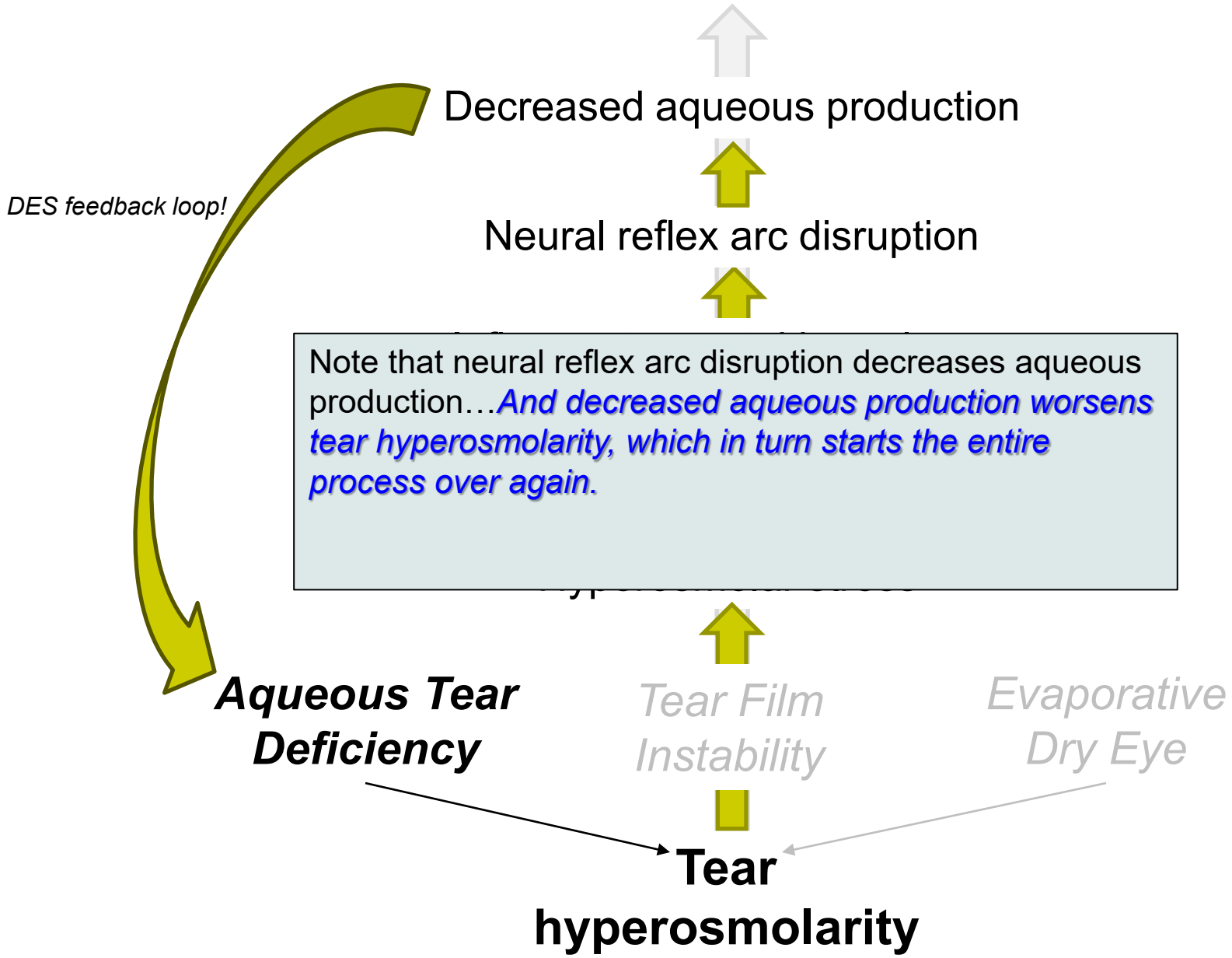
**hyperosmolarity**

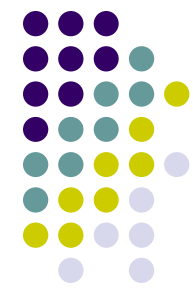




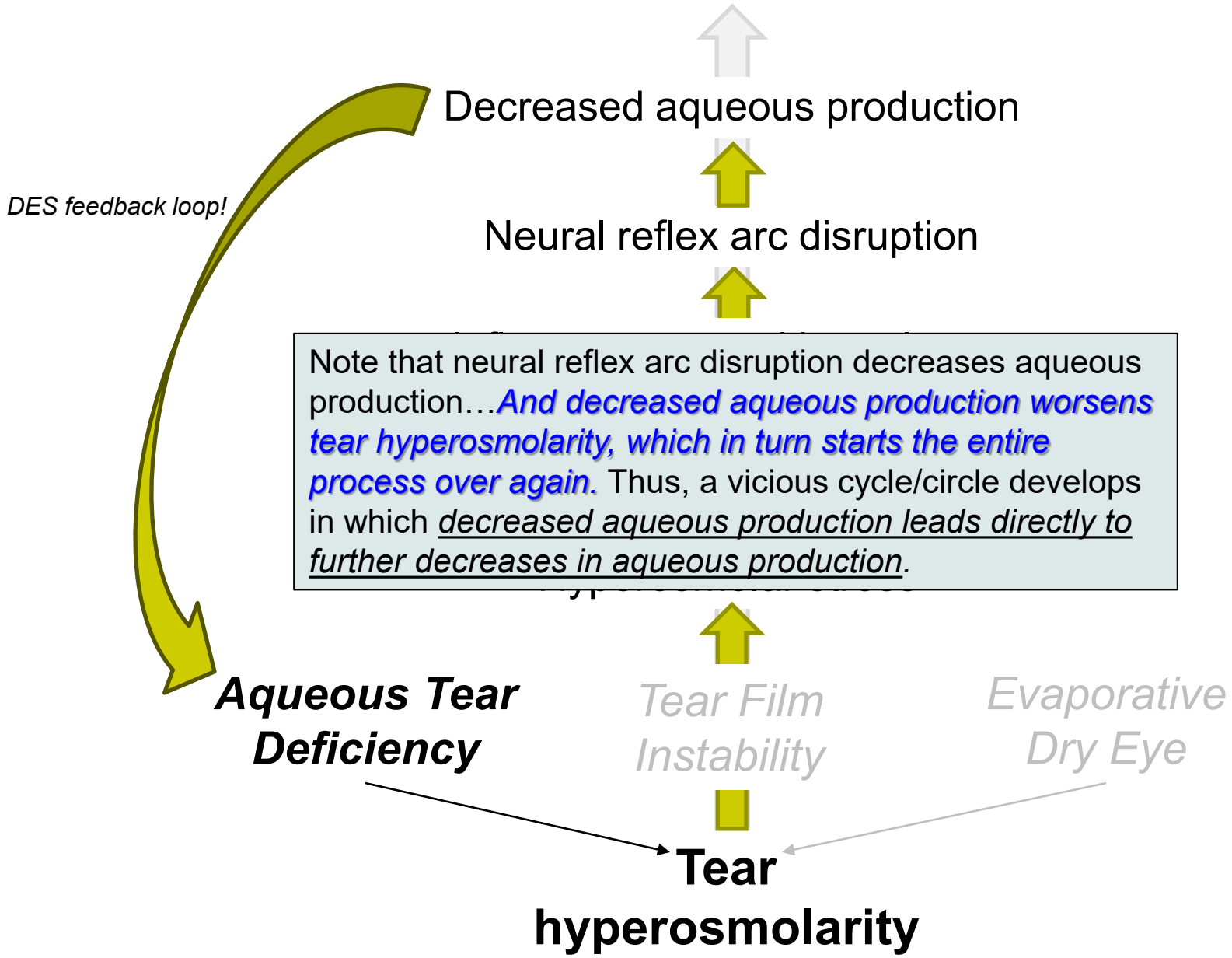


# Dry Eye Syndrome

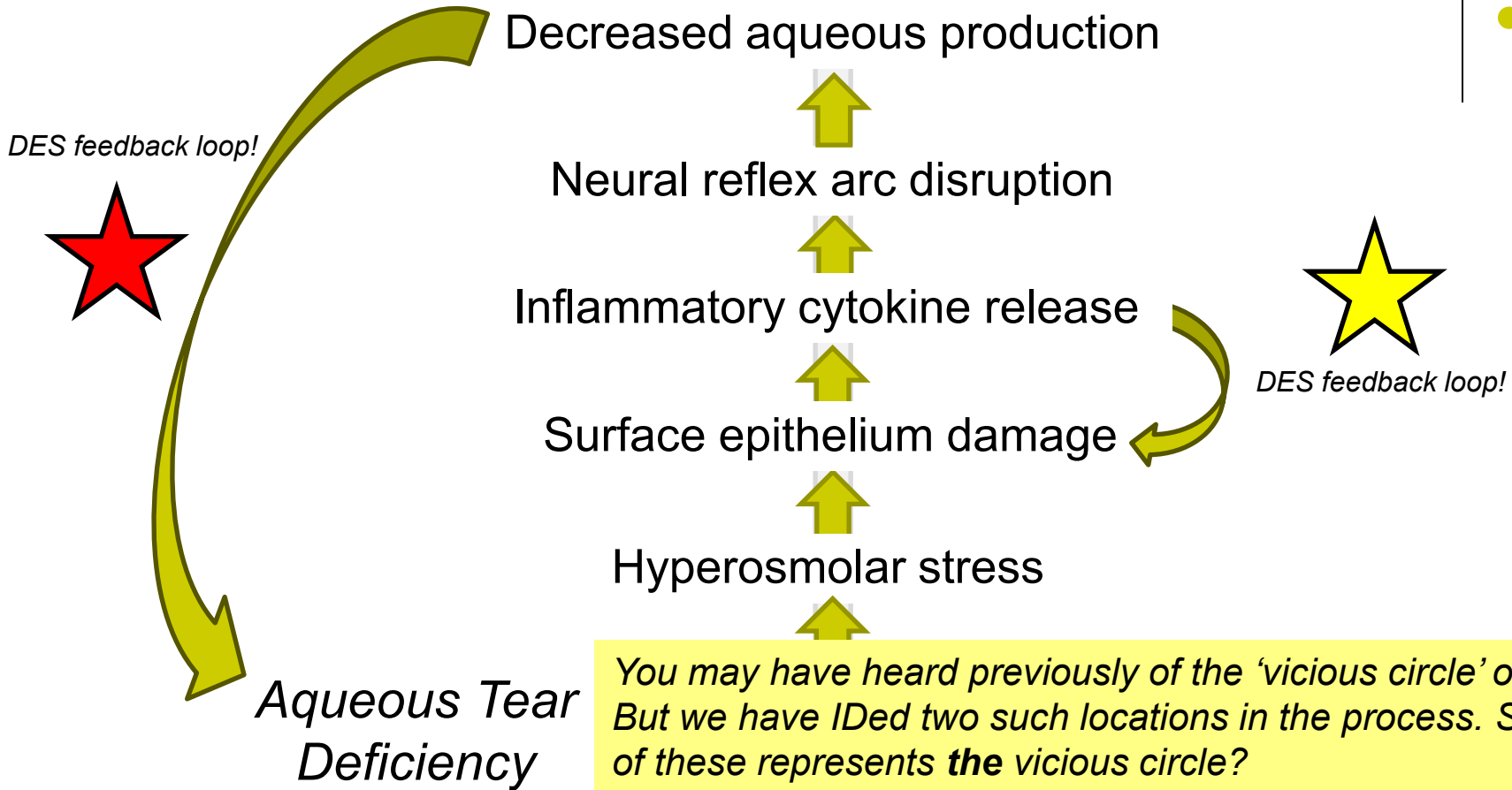




# Dry Eye Syndrome

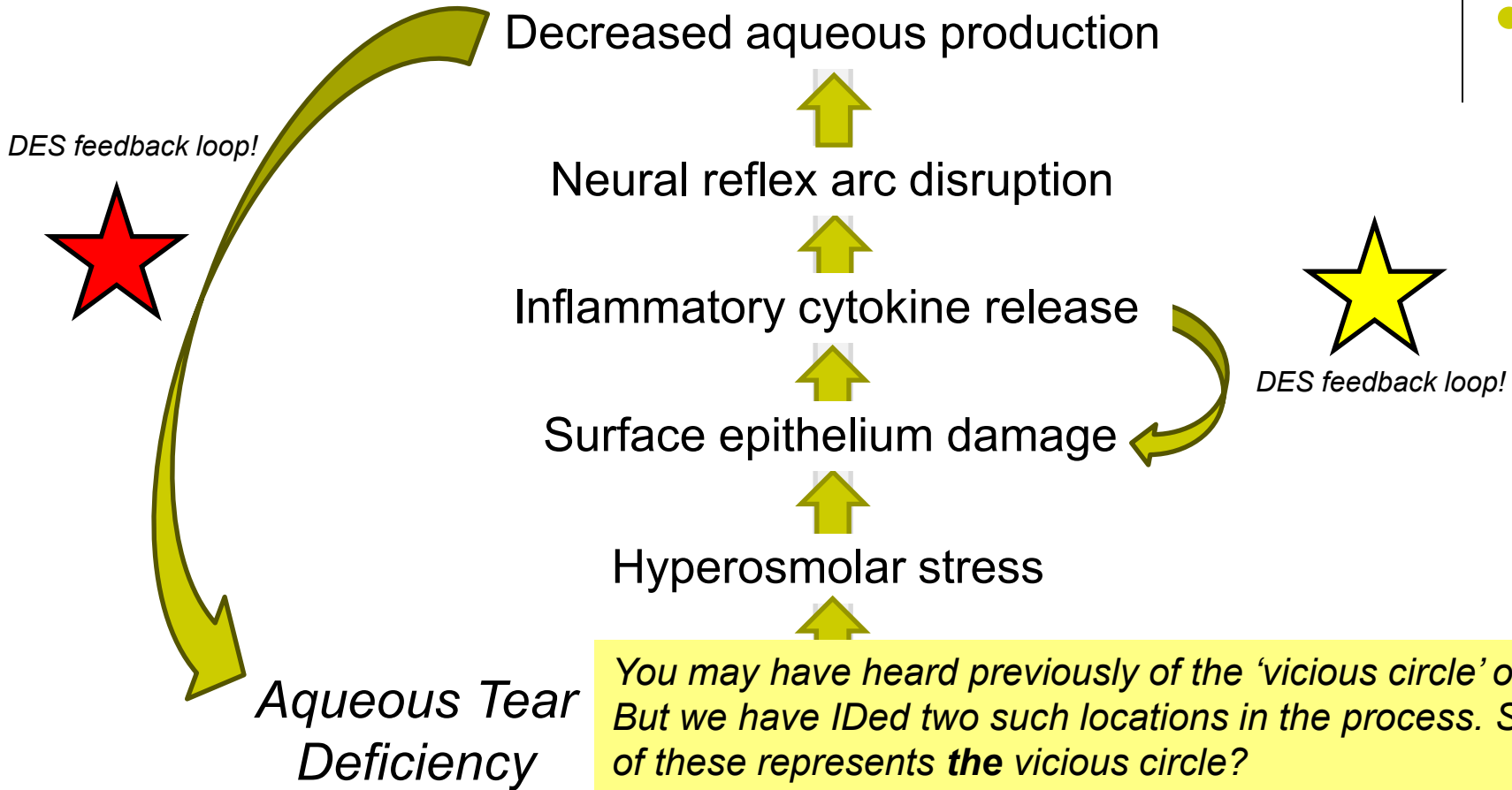
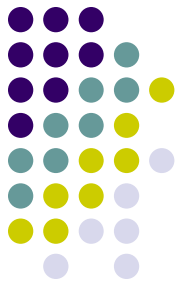


# Dry Eye Syndrome

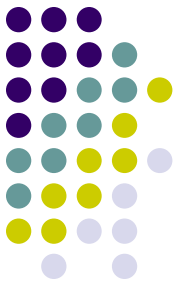


*You may have heard previously of the 'vicious circle' of DES. But we have IDed two such locations in the process. So which of these represents **the** vicious circle?*

# Dry Eye Syndrome

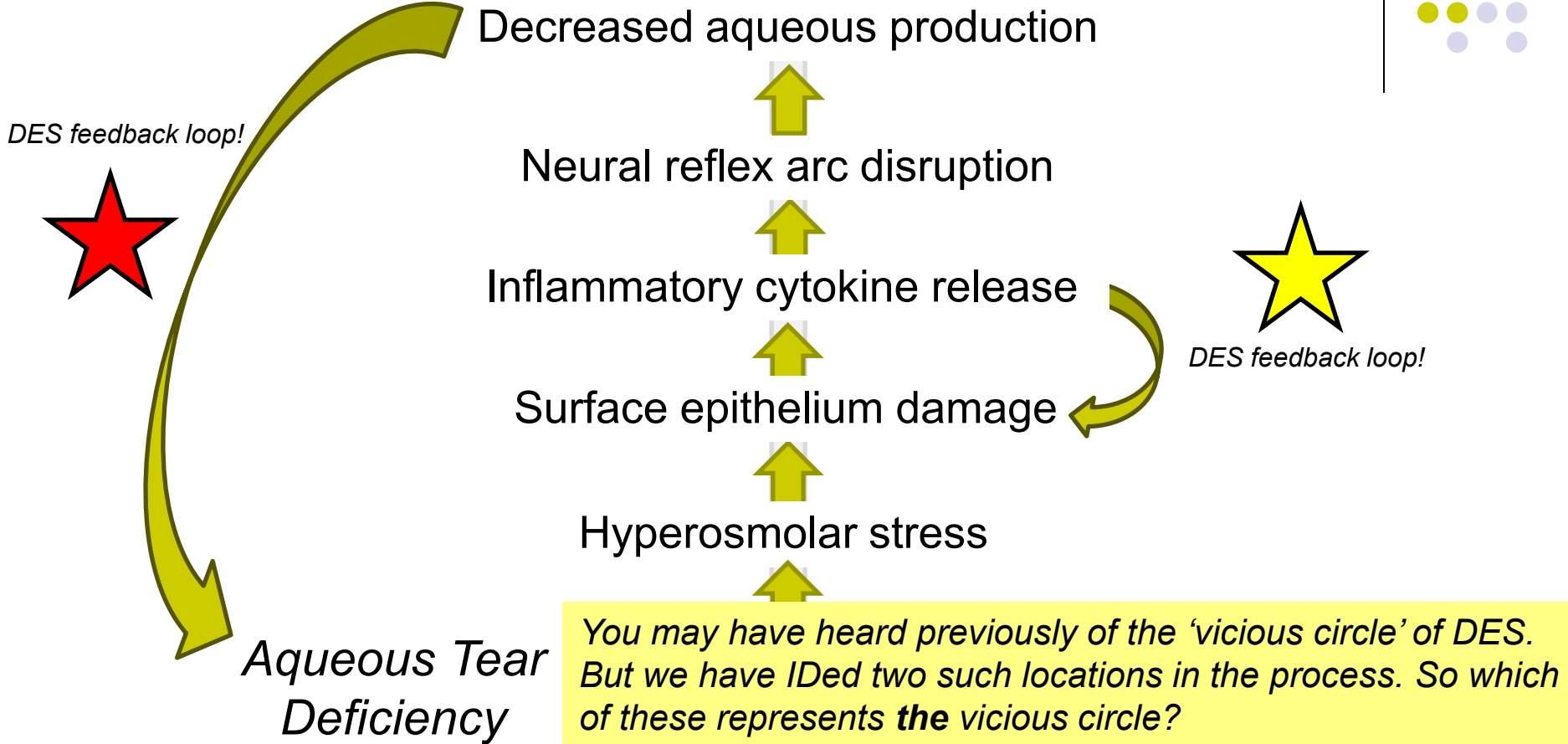


*You may have heard previously of the 'vicious circle' of DES. But we have IDed two such locations in the process. So which of these represents **the** vicious circle? That depends on who you ask, and making you aware of this dependency is the point of this question.*



# Dry Eye Syndrome

Some *Academy* sources refer to **this** as 'the' vicious cycle of DES...



*You may have heard previously of the 'vicious circle' of DES. But we have IDed two such locations in the process. So which of these represents **the** vicious circle?*

*That depends on who you ask, and making you aware of this dependency is the point of this question.*



# Dry Eye Syndrome

Some *Academy* sources refer to **this** as 'the' vicious cycle of DES...

*DES feedback loop!*



Decreased aqueous production



Neural reflex arc disruption



Inflammatory cytokine release



Surface epithelium damage



Hyperosmolar stress



*DES feedback loop!*

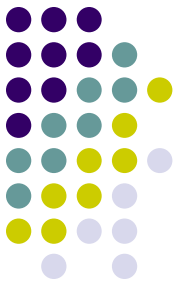


...whereas other *Academy* sources refer to **this** as such.

*Aqueous Tear Deficiency*

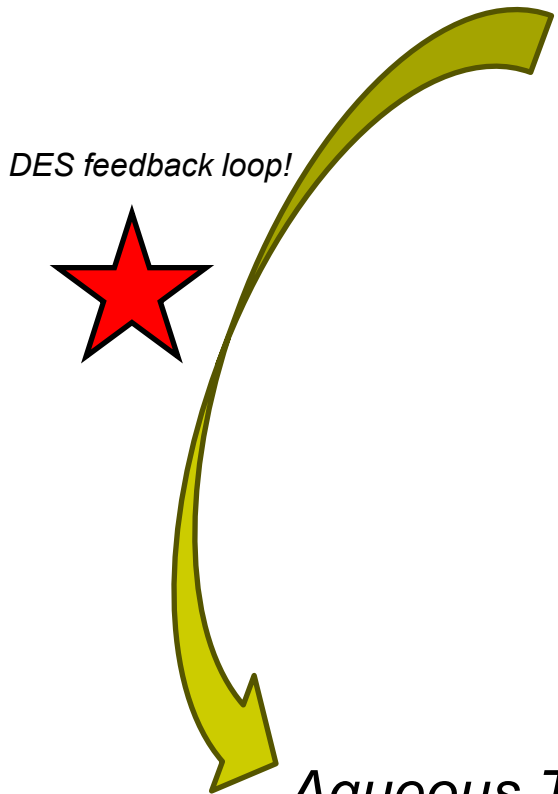
*You may have heard previously of the 'vicious circle' of DES. But we have IDed two such locations in the process. So which of these represents **the** vicious circle?*

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# Dry Eye Syndrome

Some *Academy* sources refer to **this** as 'the' vicious cycle of DES...



Decreased aqueous production



Neural reflex arc disruption



Inflammatory cytokine release



Surface epithelium damage



Hyperosmolar stress

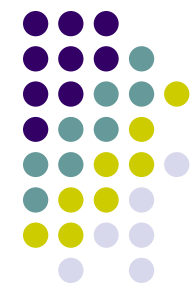


Aqueous Tear Deficiency



...whereas other *Academy* sources refer to **this** as such.

*You may have heard previously of the 'vicious circle' of DES. But we have IDed two such locations in the process. So which of these represents **the** vicious circle? That depends on who you ask, and making you aware of this dependency is the point of this question. So when getting pimped re the DES vicious circle concept, be aware your attending might have one **or** the other in mind, and so be prepared to modify your response accordingly!*

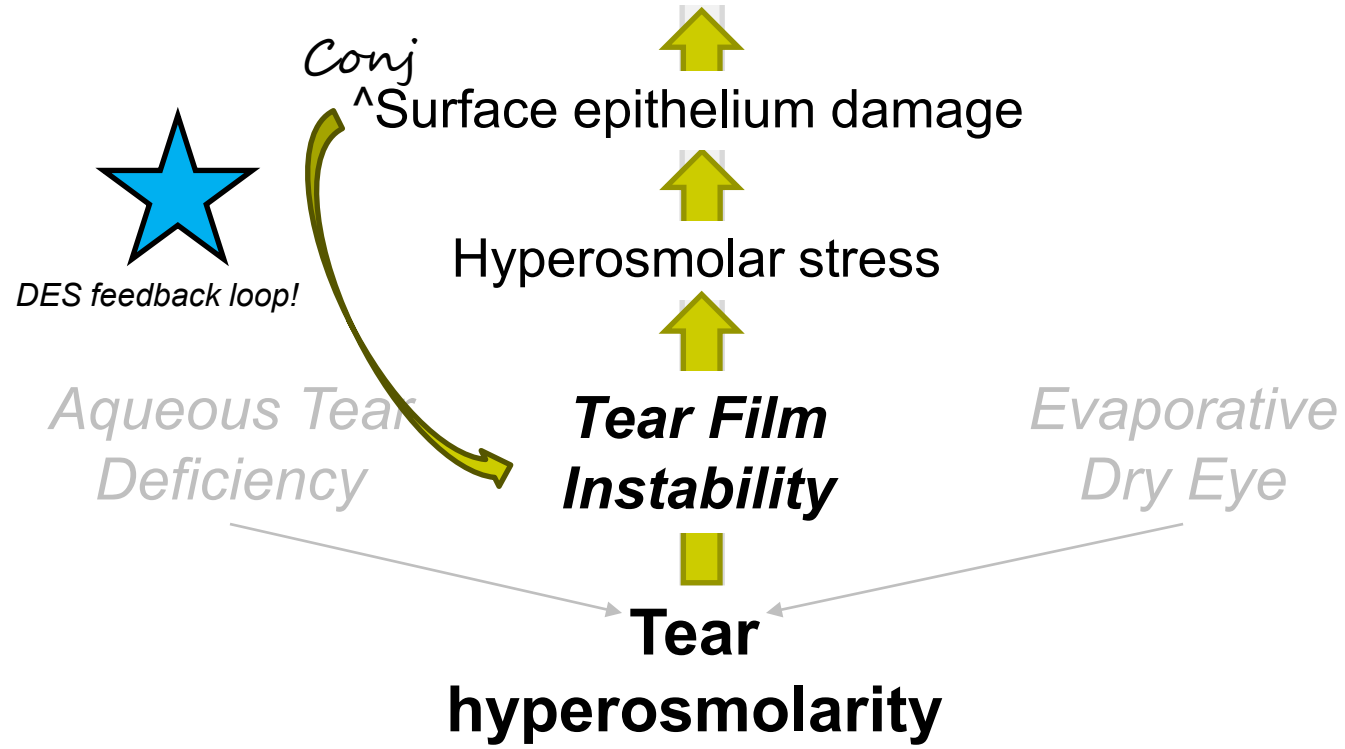


# Dry Eye Syndrome

Decreased aqueous production



*Hol up—we also identified **this** (increased TFI → hyperosmolar epi damage → decreased number of goblet cells → increased TFI) vicious circle. What about it?*



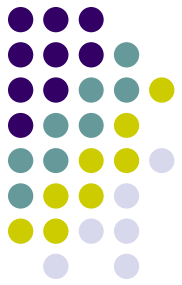
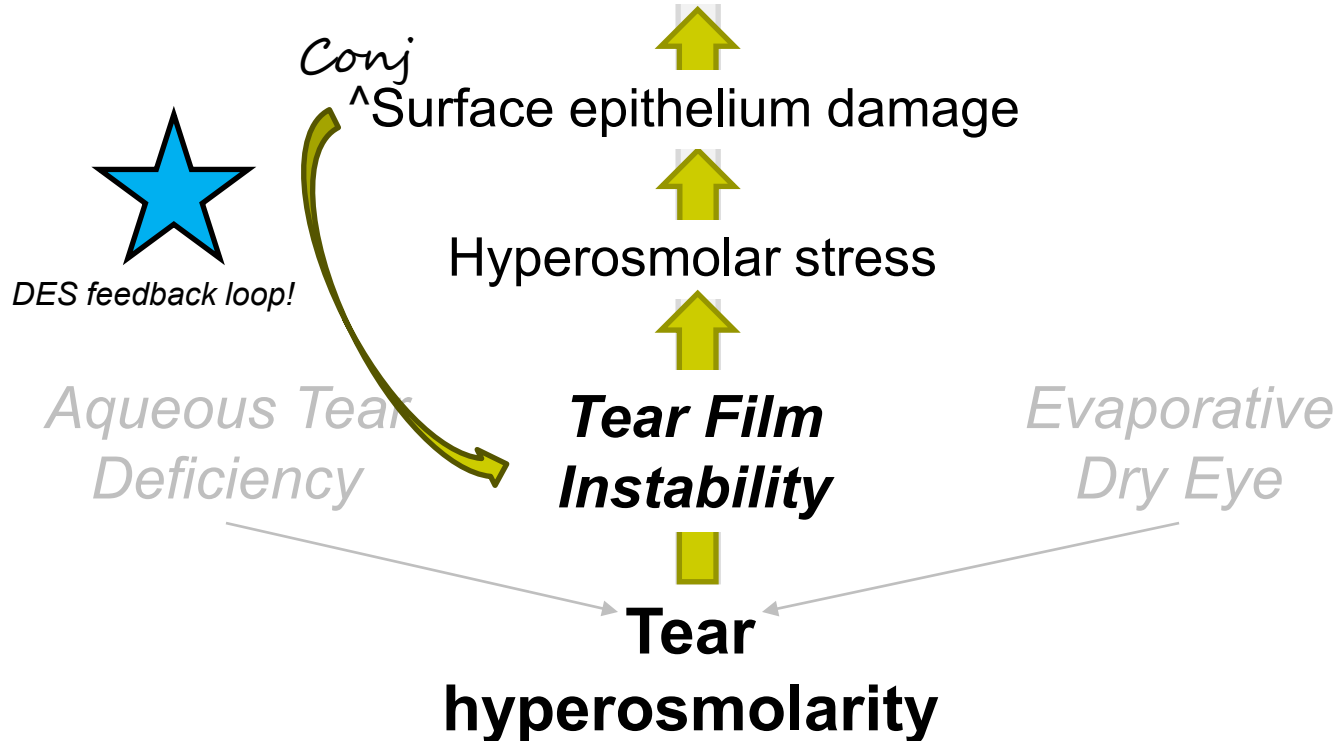


# Dry Eye Syndrome

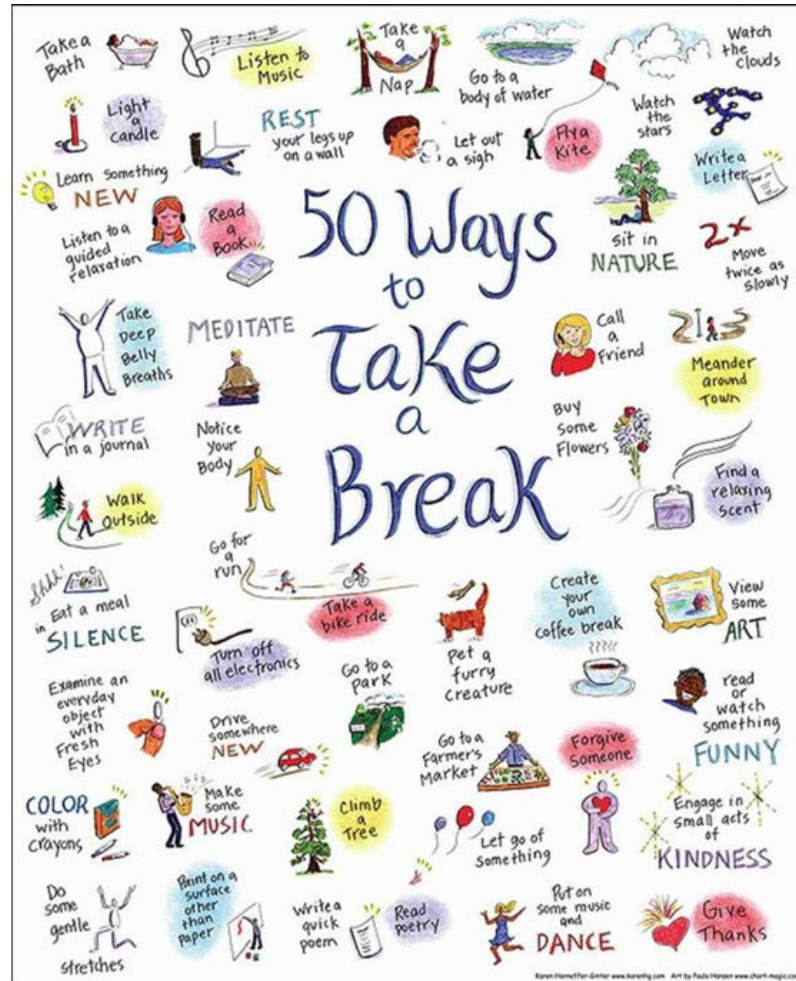
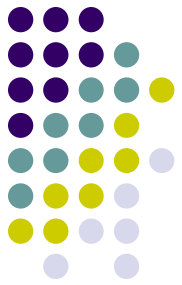
Decreased aqueous production



*Hol up—we also identified **this** (increased TFI → hyperosmolar epi damage → decreased number of goblet cells → increased TFI) vicious circle. What about it? That one seems to get no love from anyone, so I doubt your attending will have it in mind if/when she mentions the ‘vicious circle of DES’*

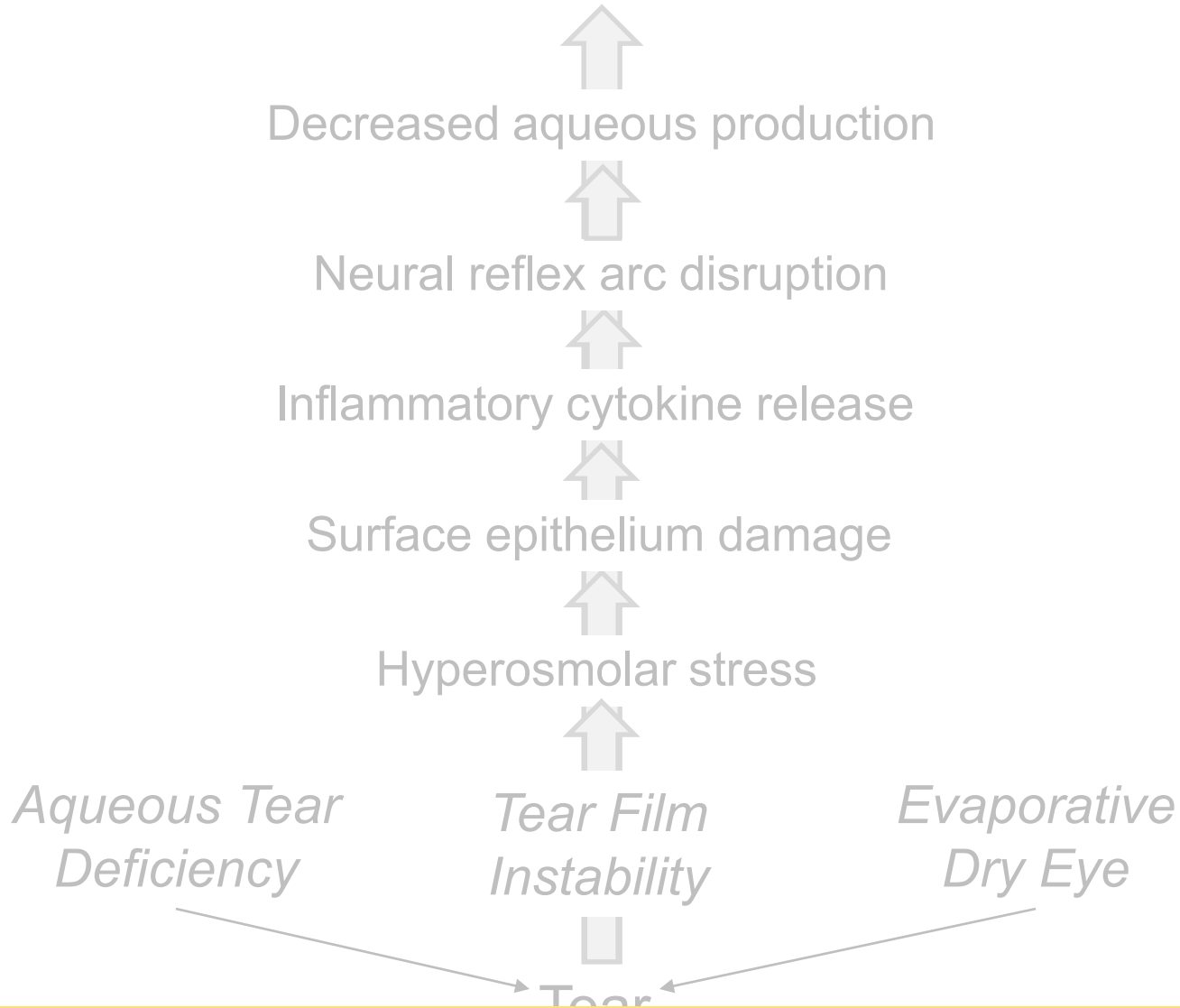


# Dry Eye Syndrome



(This is a good point in the set to take a break)

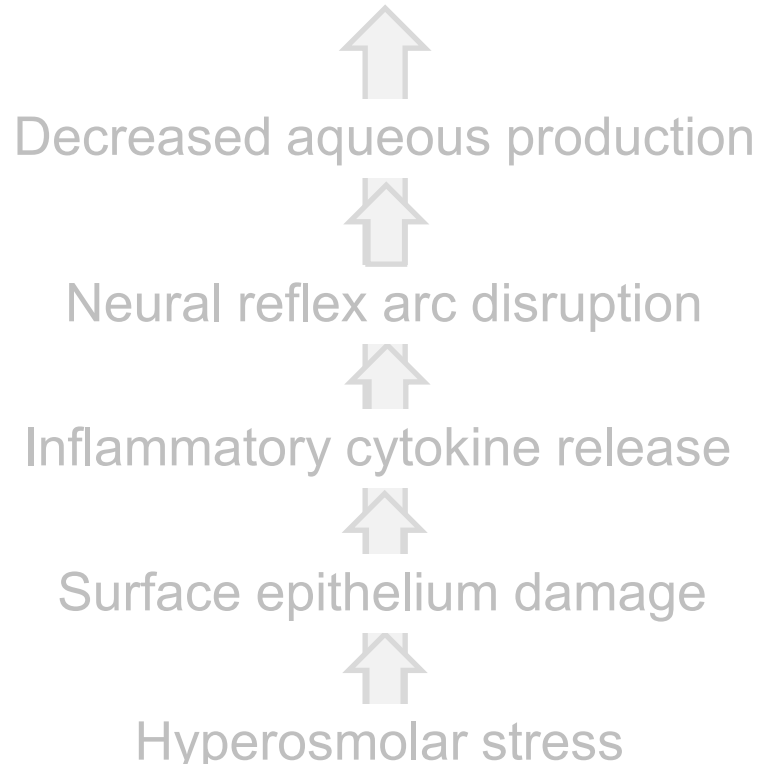
# Dry Eye Syndrome



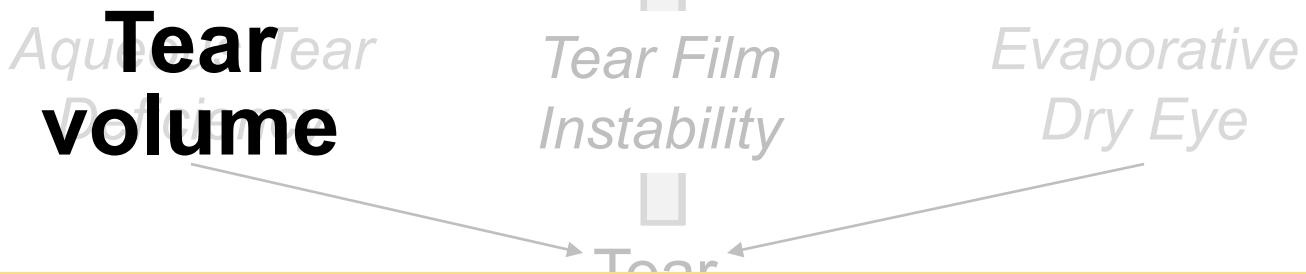
***With regards to treating DES—there are three obvious interdiction points in its pathogenesis:***



# Dry Eye Syndrome



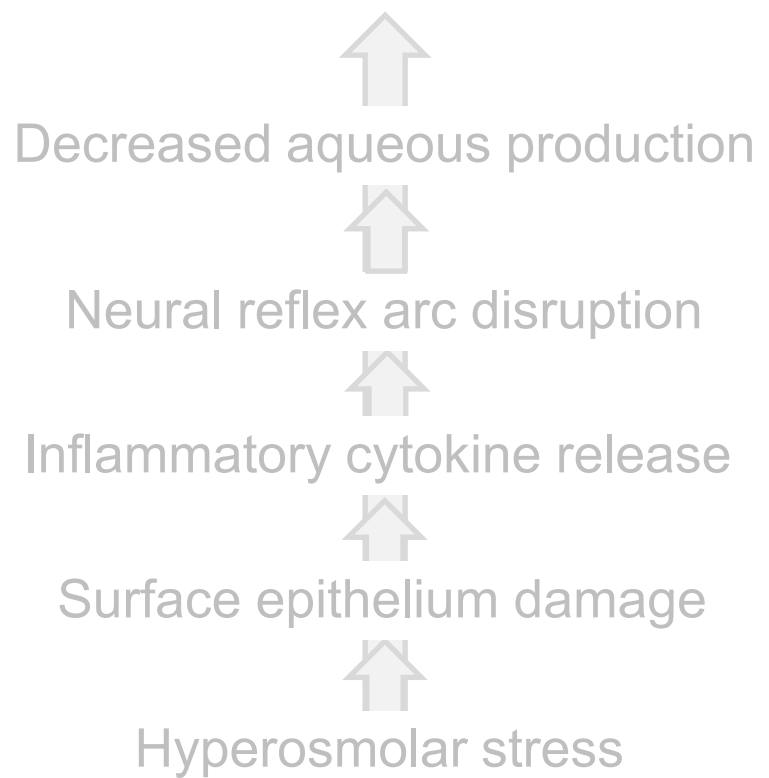
1) Increase tear volume



*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*



# Dry Eye Syndrome

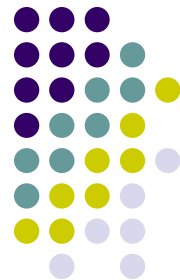


1) Increase tear volume

2) Decrease tear evaporation



*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*



# Dry Eye Syndrome

Decreased aqueous production



Neural reflex arc disruption



*3) Prevent cytokine release and/or mitigate their effects*

**Inflammatory cytokine release**



Surface epithelium damage



Hyperosmolar stress



*1) Increase tear volume*

*2) Decrease tear evaporation*



**Tear volume**

**Tear Film Instability**

**Tear evaporation**



*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*



# Dry Eye Syndrome

Decreased aqueous production

*What is the most straightforward means of increasing aqueous volume?*

*mitigate their effects*

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

1) Increase tear volume

2) Decrease tear evaporation

**Tear volume**

Tear Film Instability

Evaporative Dry Eye

*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*



# Dry Eye Syndrome

Decreased aqueous production

What is the most straightforward means of increasing aqueous volume?  
Supplementing the tear lake with two words

*mitigate their effects*

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

1) Increase tear volume

2) Decrease tear evaporation

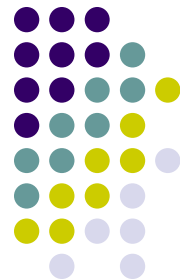
**Tear volume**

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:





# Dry Eye Syndrome



Decreased aqueous production



*What is the most straightforward means of increasing aqueous volume?  
Supplementing the tear lake with artificial tears*

*mitigate their effects*

Inflammatory cytokine release



Surface epithelium damage



Hyperosmolar stress



1) Increase tear volume

2) Decrease tear evaporation

**Tear volume**

Tear Film Instability

Evaporative Tear evaporation Dry Eye



*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*



# Dry Eye Syndrome

Decreased aqueous production

*What is the most straightforward means of increasing aqueous volume? Supplementing the tear lake with artificial tears. The Cornea book says tear substitutes are “the mainstay of treatment for ATD.”*

*mitigate their effects* Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

1) Increase tear volume

2) Decrease tear evaporation

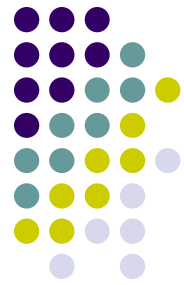
**Tear volume**

Tear Film Instability

Evaporative Tear evaporation Dry Eye

*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



Decreased aqueous production

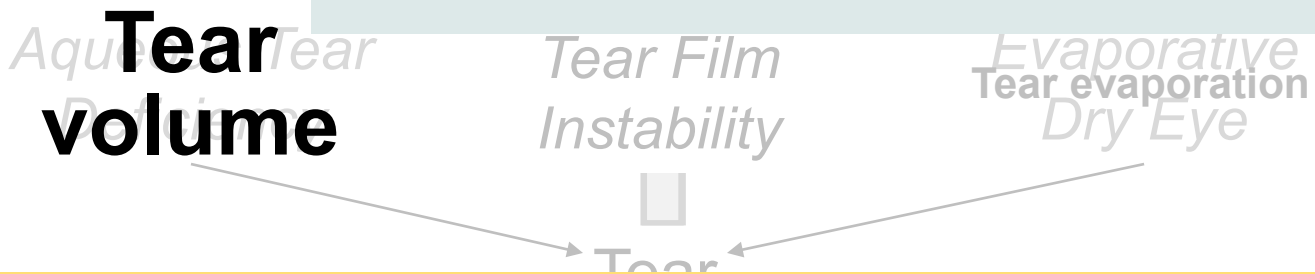


What is the most straightforward means of increasing aqueous volume?  
Supplementing the tear lake with **artificial tears**. The *Cornea* book says  
tear substitutes are “the mainstay of treatment for ATD.”

mitigate their effects

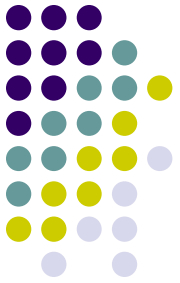
What is the formal name for artificial tears?

1) Increase tear volume



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production

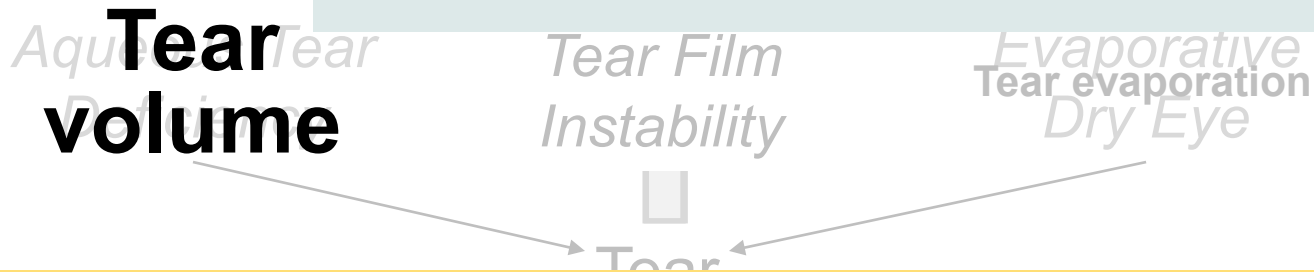


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tear substitutes are “the mainstay of treatment for ATD.”

mitigate their effects

What is the formal name for artificial tears?  
They are ophthalmic

1) Increase tear volume



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production

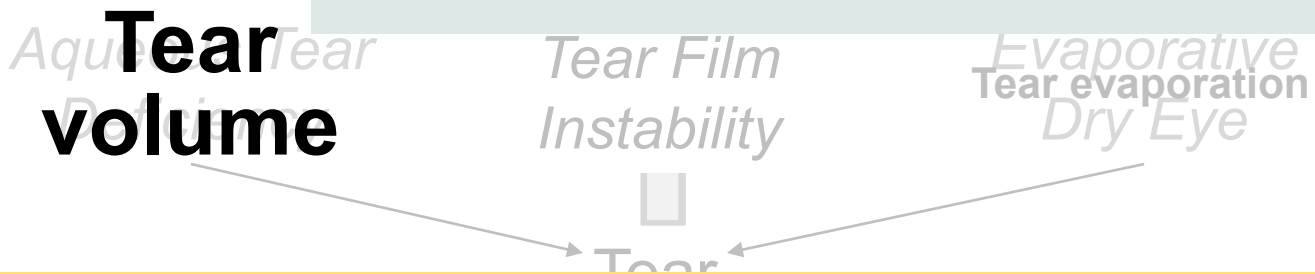


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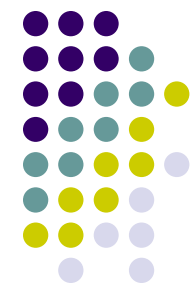
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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



Decreased aqueous production



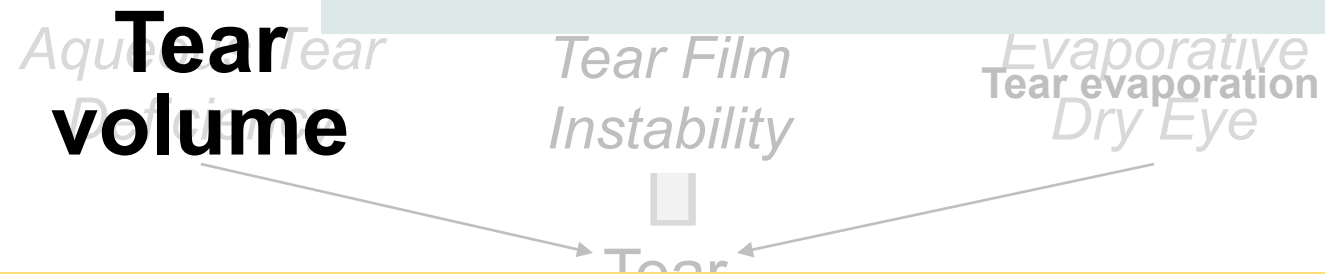
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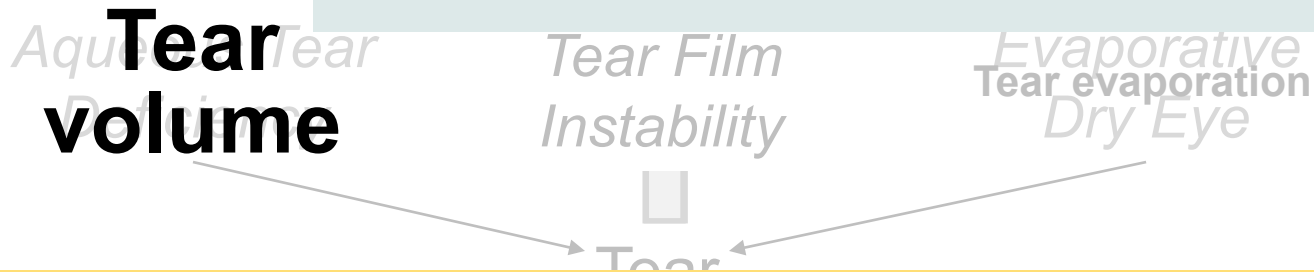
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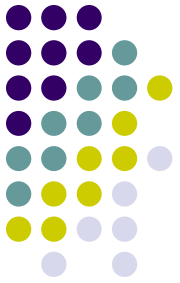
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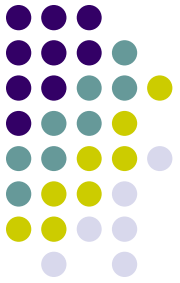
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Aqueous Tear  
Production  
**Tear  
volume**

Tear Film  
Instability

Evaporative  
Tear evaporation  
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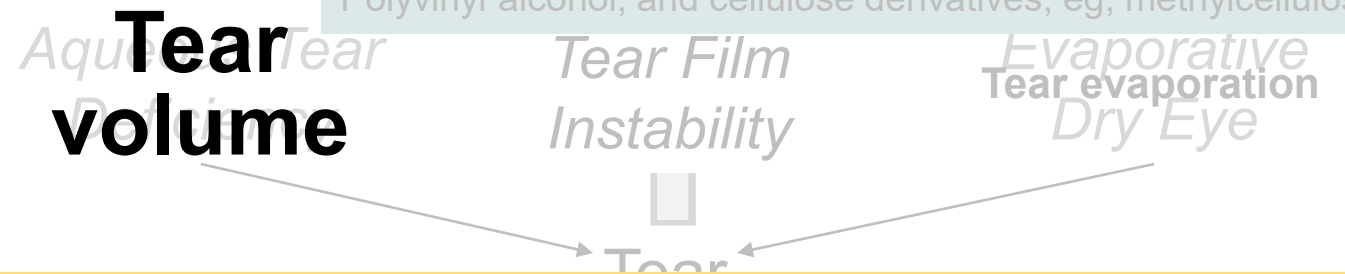
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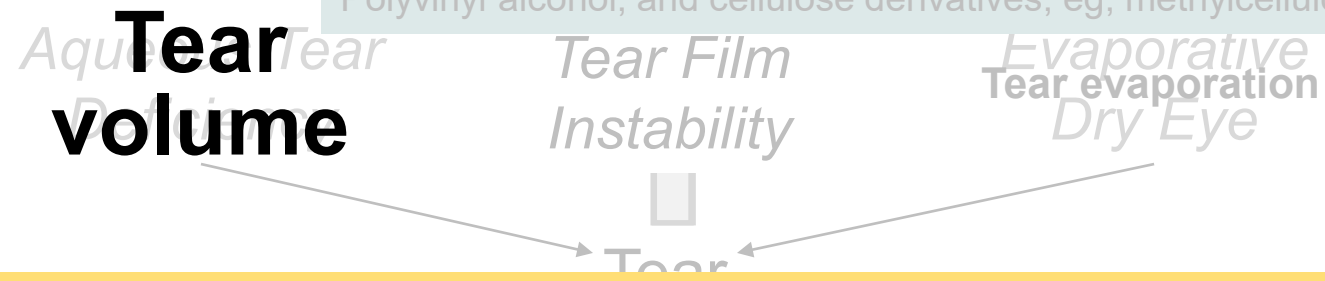


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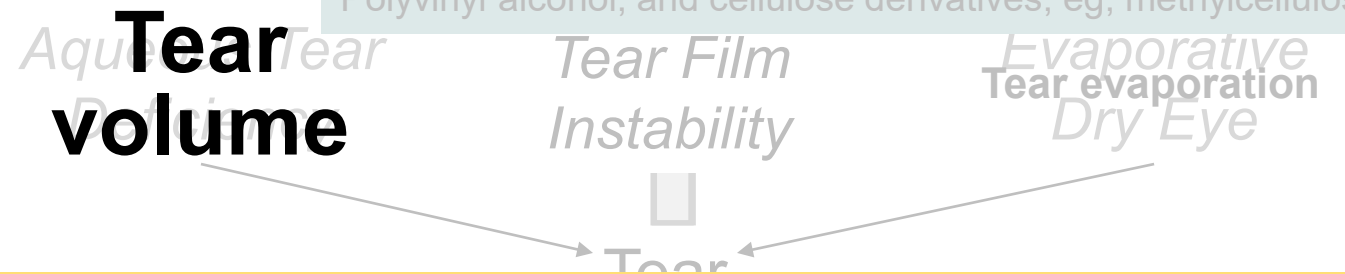
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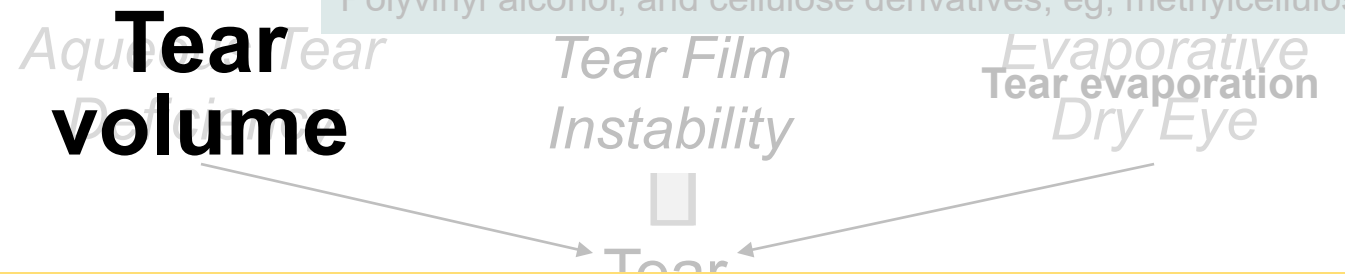
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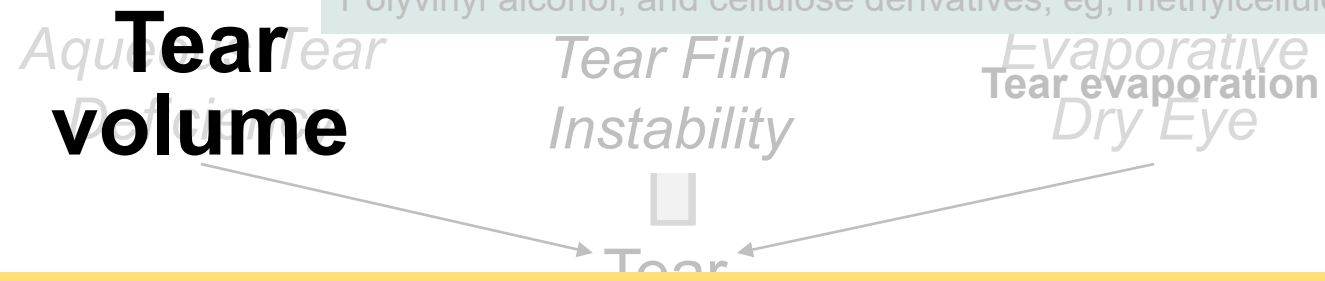
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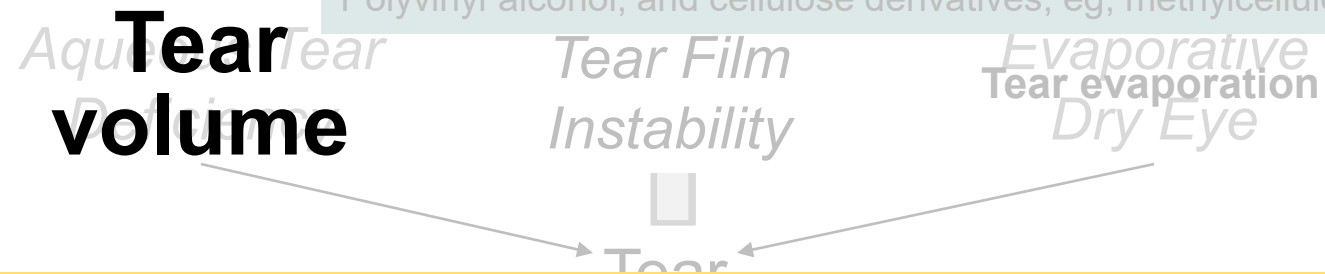
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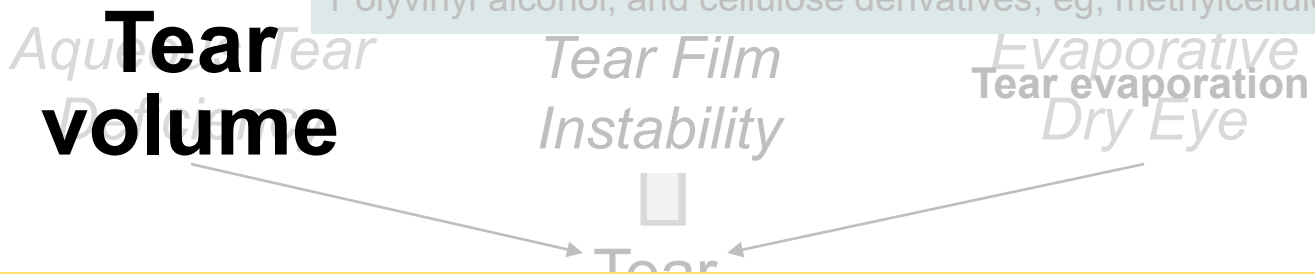
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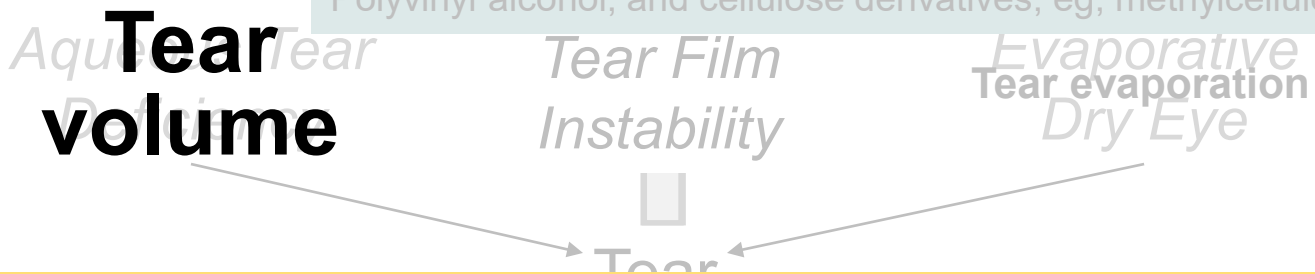
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membranes  
at conveys  
are the

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**Tear volume**

Instability

tear evaporation  
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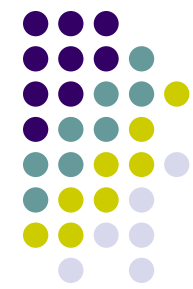
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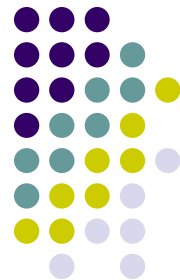
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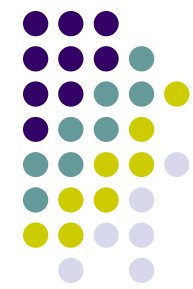
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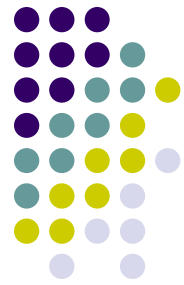
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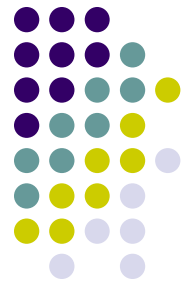


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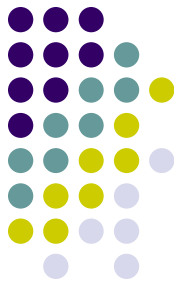


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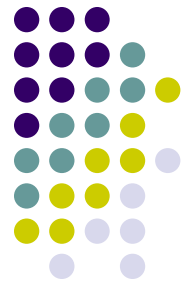
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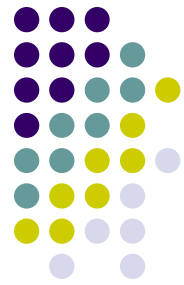
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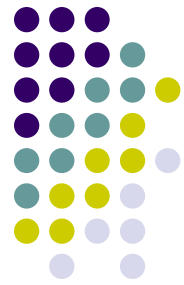
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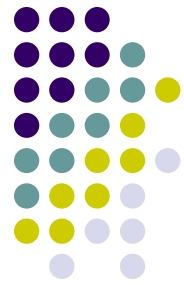


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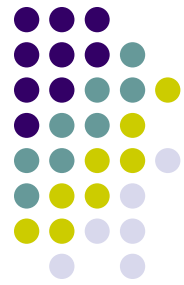
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Dry Eye



Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



What complications are associated with punctal occlusion?  
--?  
--?

What is the...  
Supplementing...  
tear substitute...  
mitigate the

There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

There are two general ways to occlude the puncta—what are they? Briefly, what is involved in each?

Reversible and permanent. In *reversible occlusion*, a plug (composed of silicone, usually) is stuffed into the punctum, blocking it. *Permanent occlusion* involves applying heat to the inner aspect of the punctum, scarring it closed.

membranes  
at conveys  
it are the

1) Increase

available drops, so if a pt c/o stinging tell them to dry a different formulation.

**Tear volume**

Instability

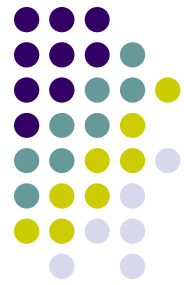
tear evaporation  
Dry Eye



Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



What complications are associated with punctal occlusion?  
--They can fail: Inserts can be dislodged; adhesions can open up  
--?

What is the...  
Supplementing...  
tear substitute...  
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There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

There are two general ways to occlude the puncta—what are they? Briefly, what is involved in each?

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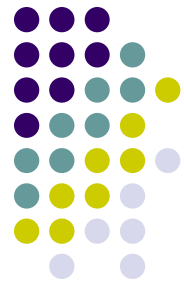
tear evaporation  
Dry Eye



Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



*What complications are associated with punctal occlusion?*  
--They can fail: Inserts can be dislodged; adhesions can open up  
--Inserts can be over inserted, ie, into the [redacted]

*What is the [redacted]  
Supplementing [redacted]  
tear substitute [redacted]  
mitigate the [redacted]*

There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

*There are two general ways to occlude the puncta—what are they? Briefly, what is involved in each?*  
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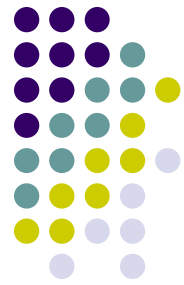
Instability

tear evaporation  
Dry Eye



*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



*What complications are associated with punctal occlusion?*  
--They can fail: Inserts can be dislodged; adhesions can open up  
--Inserts can be over inserted, ie, into the canaliculus

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Supplementing...  
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There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

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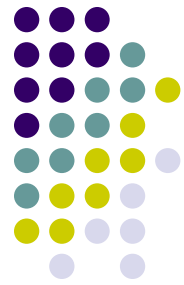
tear evaporation  
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Tear

*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



*What complications are associated with punctal occlusion?*  
--They can fail: Inserts can be dislodged; adhesions can open up  
--Inserts can be over inserted, ie, into the canaliculus, which means surgical removal will be required if inflammation or infection develops

*What is the...  
Supplementing...  
tear substitute...  
mitigate the...*

There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

*There are two general ways to occlude the puncta—what are they? Briefly, what is involved in each?*  
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**Tear volume**

*Instability*

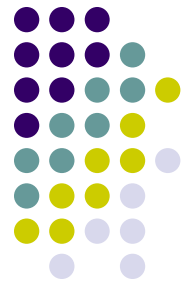
*tear evaporation  
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Tear

*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



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What is the...  
Supplementing...  
tear substitute...  
mitigate the...

There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

**Note: There is another complication induced by the use of punctal occlusion that we will cover later in the slide-set**

(composed of silicone, usually) is stuffed into the punctum, blocking it. *Permanent occlusion* involves applying heat to the inner aspect of the punctum, scarring it closed.

1) Increase

available drops, so if a pt c/o stinging tell them to dry a different formulation.

**Tea**  
**volume**

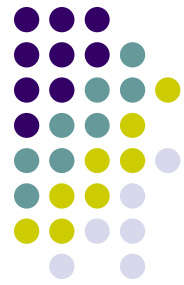
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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



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What is the...  
Supplementing...  
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There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

The Cornea book mentions two other surgical interventions—vastly less common than punctal plugging—that are occasionally indicated. What are they?  
--?  
--?

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available drops, so if a pt c/o stinging tell them to dry a different formulation.

**Tear volume**

Instability

tear evaporation  
Dry Eye

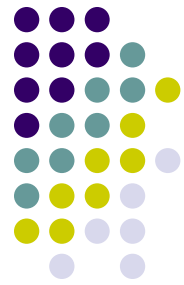


Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



What complications are associated with punctal occlusion?  
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--Inserts can be over inserted, ie, into the canaliculus, which means surgical removal will be required if inflammation or infection develops

What is the...  
Supplementing...  
tear substitute...  
mitigate the

There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

The Cornea book mentions two other surgical interventions—vastly less common than punctal plugging—that are occasionally indicated. What are they?  
--Correction of two words  
--?

(composed of silicone, usually) is stuffed into the punctum, blocking it. *Permanent occlusion* involves applying heat to the inner aspect of the punctum, scarring it closed.

1) Increase

available drops, so if a pt c/o stinging tell them to dry a different formulation.

**Tea**  
**volume**

Instability

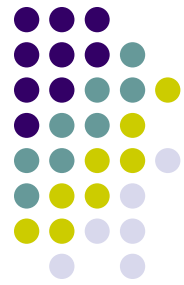
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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



What complications are associated with punctal occlusion?  
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What is the...  
Supplementing...  
tear substitute...  
mitigate the...

There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

The Cornea book mentions two other surgical interventions—vastly less common than punctal plugging—that are occasionally indicated. What are they?  
--Correction of eyelid malposition  
--?

(composed of silicone, usually) is stuffed into the punctum, blocking it. *Permanent occlusion* involves applying heat to the inner aspect of the punctum, scarring it closed.

1) Increase

available drops, so if a pt c/o stinging tell them to dry a different formulation.

**Tear volume**

Instability

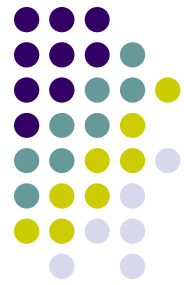
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Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



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What is the...  
Supplementing...  
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There is indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD

The Cornea book mentions two other surgical interventions—vastly less common than punctal plugging—that are occasionally indicated. What are they?  
--Correction of eyelid malposition  
--Tarsorrhaphy

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available drops, so if a pt c/o stinging tell them to dry a different formulation.

**Tea**  
**volume**

Instability

tear evaporation  
Dry Eye



Tea

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects

Inflammatory cytokine release

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

Hyperosmolar stress

1) Increase tear volume

2) Decrease tear evaporation

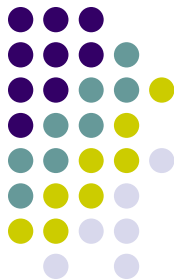
Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



3) Prevent cytokine release and/or mitigate their effects

Inflammatory cytokine release



Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?  
Lid hygiene. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

hyperosmolar stress

1) Increase tear volume

2) Decrease tear evaporation



Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye



Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



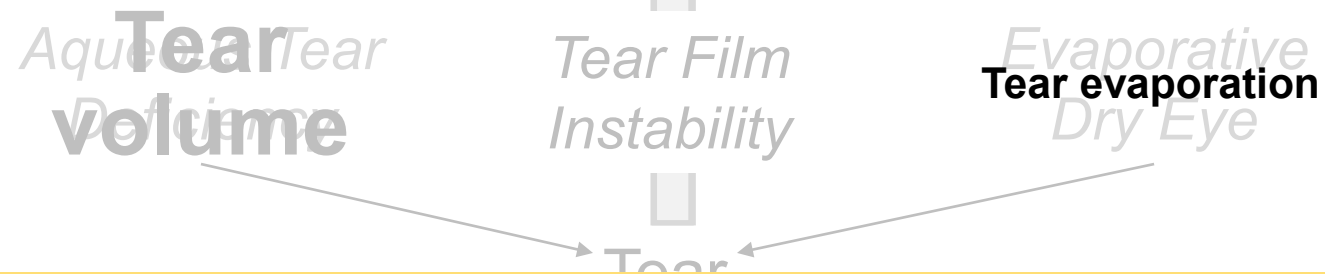
3) Prevent cytokine release and/or mitigate their effects

Just as ATs are mainstay tx of  
**Lid hygiene**  
part [of tx] at a

What two fundamental steps are involved in lid hygiene?  
1) ?  
2) ?

1) Increase tear volume

2) Decrease tear evaporation



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



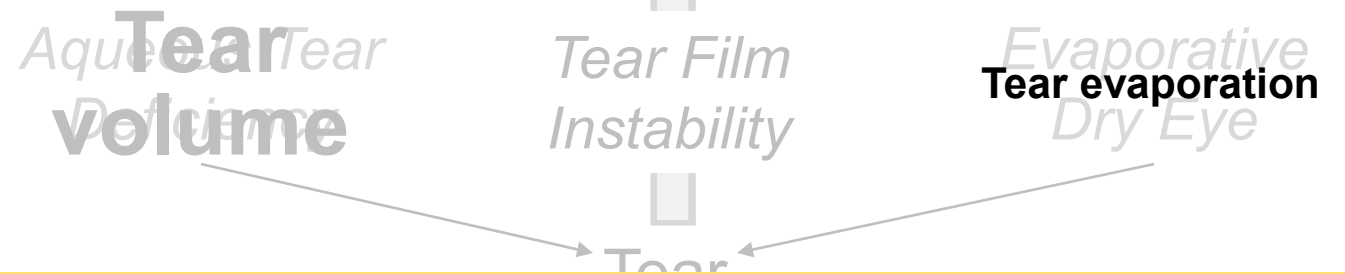
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Just as ATs are mainstay tx of  
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What two fundamental steps are involved in lid hygiene?  
 1) Application of [ ] to the eyelids to [ ] the abnormal meibum  
 2) ?

1) Increase tear volume

2) Decrease tear evaporation



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects Inflammatory cytokine release

Just as ATs are mainstay tx of Lid hygiene

What two fundamental steps are involved in lid hygiene?  
1) Application of heat to the eyelids to soften the abnormal meibum  
2) ?

1) Increase tear volume

2) Decrease tear evaporation

Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:





# Dry Eye Syndrome



3) Prevent cytokine release and/or mitigate their effects

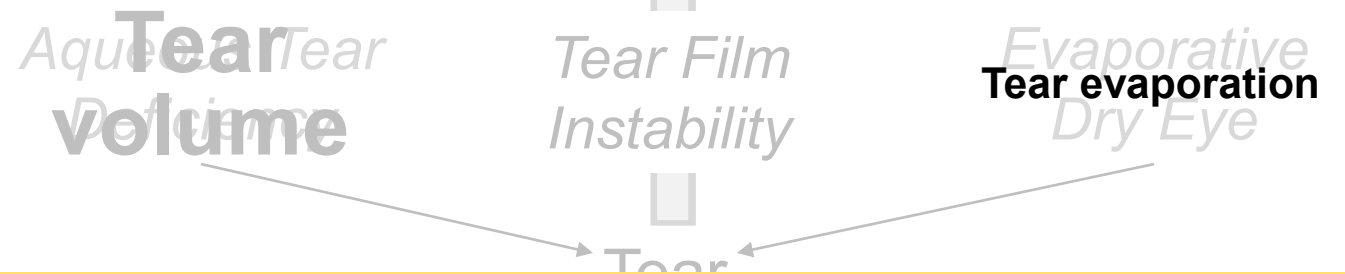
Lid hygiene

What two fundamental steps are involved in lid hygiene?

- 1) Application of heat to the eyelids to soften the abnormal meibum
- 2) [ ] of the lid margin to [ ] the abnormal meibum

1) Increase tear volume

2) Decrease tear evaporation



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

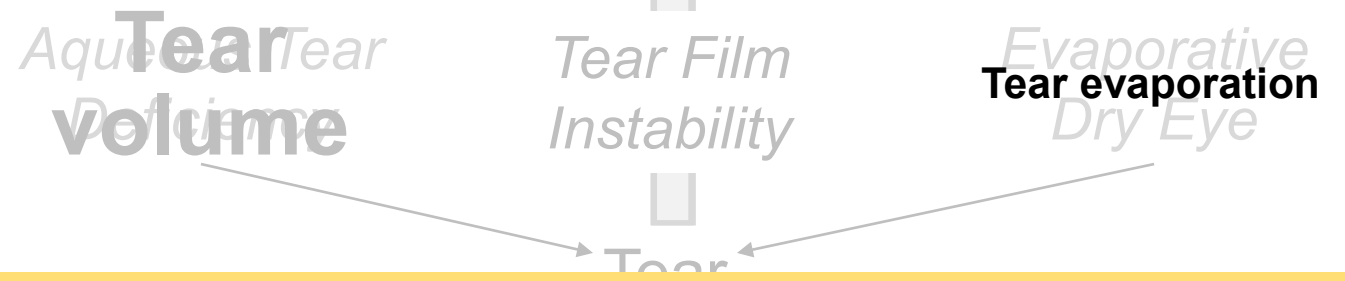
3) Prevent cytokine release and/or mitigate their effects Inflammatory cytokine release

Just as ATs are mainstay tx of Lid hygiene

What two fundamental steps are involved in lid hygiene?  
1) Application of heat to the eyelids to soften the abnormal meibum  
2) Compression/massage of the lid margin to express the abnormal meibum

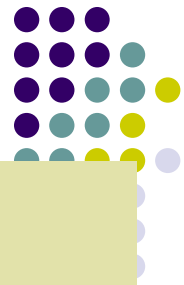
1) Increase tear volume

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



What does it mean to say meibum is 'abnormal'?

3) Prevent or mitigate the

mainstay tx of 1) Application of heat to the eyelids to soften the  
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2) Compression/massage of the lid margin to express part [of tx] at a

Lid hygiene

abnormal meibum

1) Increase tear volume

2) Decrease tear evaporation

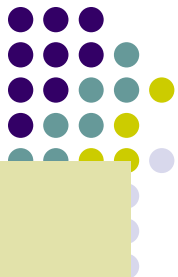
Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



What does it mean to say meibum is 'abnormal'?  
It means its chemical composition has been altered (and not for the better)

3) Prevent or mitigate the

**Lid hygiene**      **abnormal meibum**

1) Increase tear volume

2) Decrease tear evaporation

Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



*What does it mean to say meibum is 'abnormal'?*  
It means its chemical composition has been altered (and not for the better)

*What are the knock-on effects of this chemical abnormality?*

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2) Decrease tear evaporation

Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



*What does it mean to say meibum is 'abnormal'?*  
It means its chemical composition has been altered (and not for the better)

*What are the knock-on effects of this chemical abnormality?*  
There are several, but chief among them is they induce a change in the two words of meibum

3) Prevent or mitigate the

**Lid hygiene** **abnormal meibum**

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Aqueous Tear Volume



Tear Film Instability

2) Decrease tear evaporation

Evaporative Dry Eye

**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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3) Prevent or mitigate the

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Evaporative Dry Eye

**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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3) Prevent or mitigate the

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Lid hygiene

abnormal meibum

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Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



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There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a solid v liquid at body temperature

3) Prevent or mitigate the

mainstay tx of **abnormal meibum**

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Aqueous Tear Volume

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Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

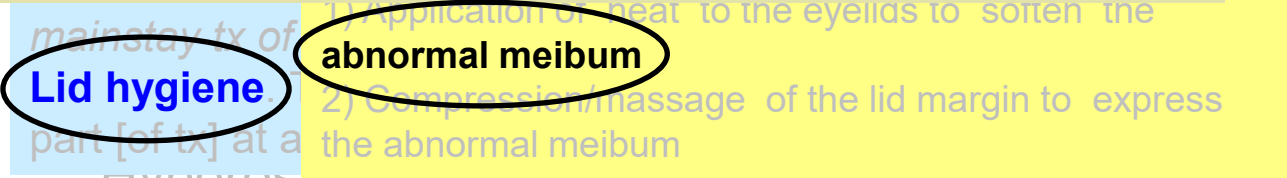
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3) Prevent or mitigate the



1) Increase tear volume

2) Decrease tear evaporation

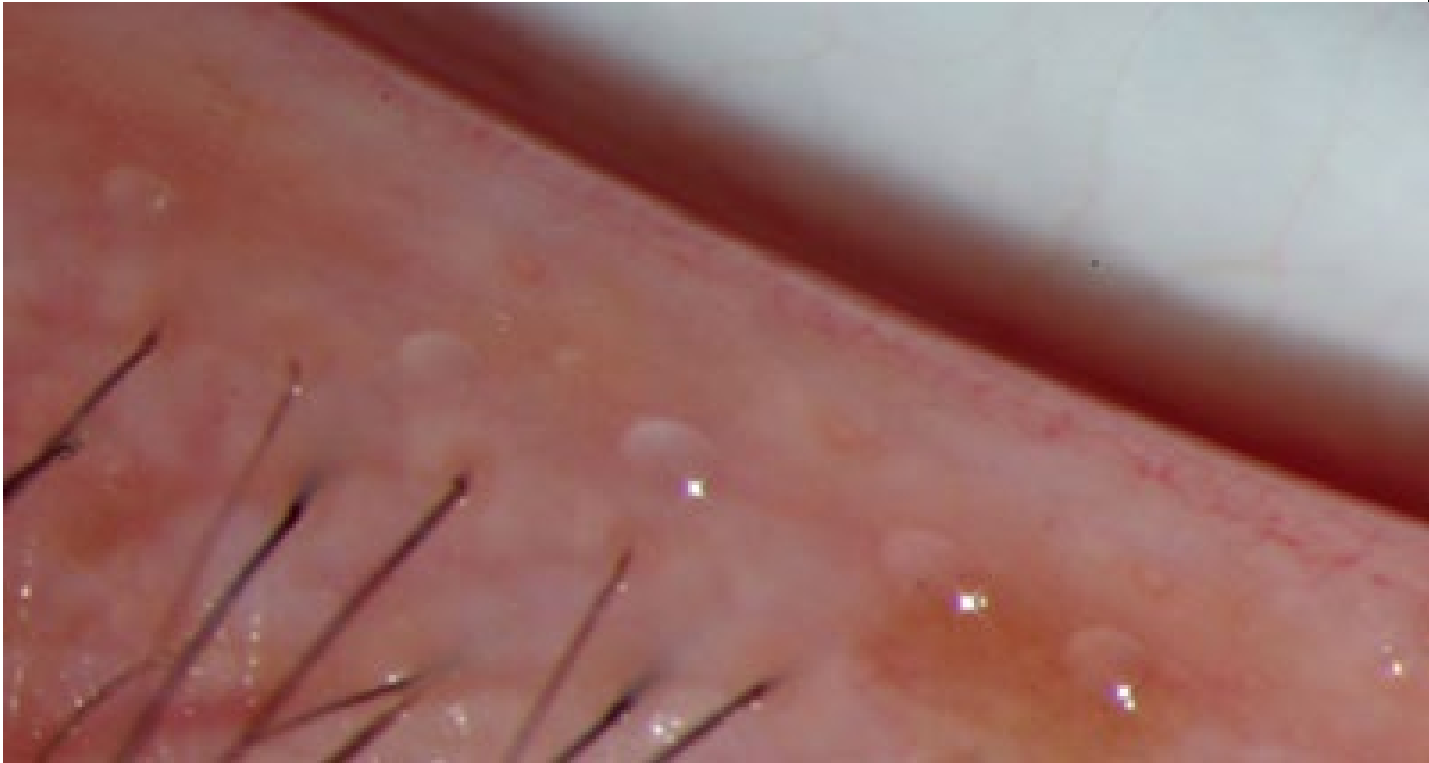
Aqueous Tear Volume

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Evaporative Dry Eye

**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



Normal meibum

# Dry Eye Syndrome



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*What are the knock-on effects of this chemical abnormality?*  
 There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a solid at body temperature

3) Prevent or mitigate the

solid v liquid

mainstay tx of **abnormal meibum**

1) Application of heat to the eyelids to soften the  
 2) Compression/massage of the lid margin to express part [of tx] at a

**Lid hygiene**

1) Increase tear volume

2) Decrease tear evaporation

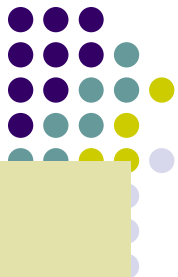
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mainstay tx of **abnormal meibum**

1) Application of heat to the eyelids to soften the  
2) Compression/massage of the lid margin to express part [of tx] at a

1) Increase tear volume

Aqueous Tear Volume



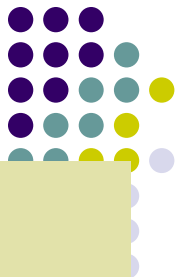
Tear Film Instability

2) Decrease tear evaporation

Evaporative Dry Eye

**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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3) Prevent or mitigate the

mainstay tx of abnormal meibum

1) Application of heat to the eyelids to soften the  
2) Compression/massage of the lid margin to express part [of tx] at a

Lid hygiene

abnormal meibum

1) Increase tear volume

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Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

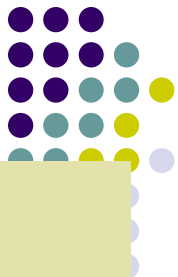
# Dry Eye Syndrome



*Ewwwww*



# Dry Eye Syndrome



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 It means its chemical composition has been altered (and not for the better)

*What are the knock-on effects of this chemical abnormality?*  
 There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

3) Prevent or mitigate the

mainstay tx of **abnormal meibum**

1) Application of heat to the eyelids to soften the  
 2) Compression/massage of the lid margin to express part [of tx] at a

1) Increase tear volume

2) Decrease tear evaporation

Aqueous Tear Volume

Tear Film Instability

Evaporative Dry Eye

**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



*What does it mean to say meibum is 'abnormal'?*  
 It means its chemical composition has been altered (and not for the better)

*What are the knock-on effects of this chemical abnormality?*  
 There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

3) Prevent or mitigate the

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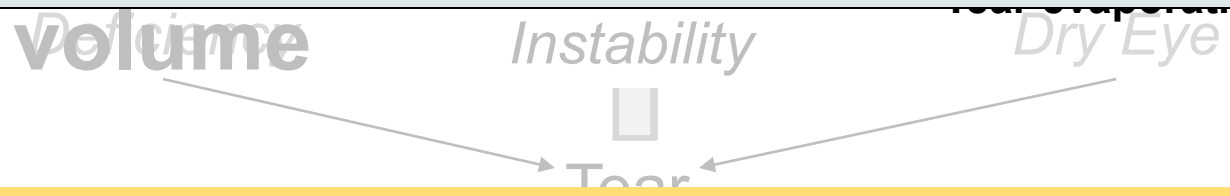
2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum

**Lid hygiene**

So the logic underpinning lid hygiene is:

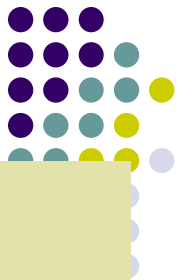
- Step 1:** Liquify the semisolid abnormal meibum clogging the glands
- Step 2:** Express the now-liquefied abnormal meibum from the glands

1) Increase

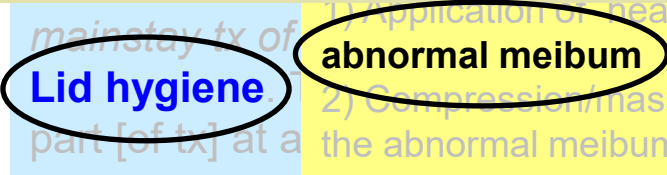


**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?



1) Application of heat to the eyelids to soften the  
2) Compression/massage of the lid margin to express part [of tx] at a

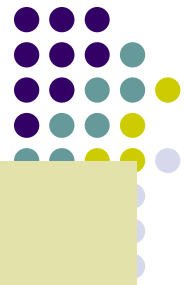
1) Increase

So the logic underpinning lid hygiene is:  
--Step 1: Liquify the semisolid abnormal meibum clogging the glands  
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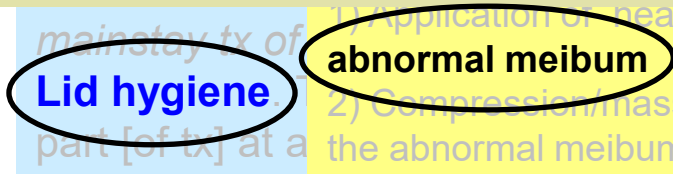


With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome

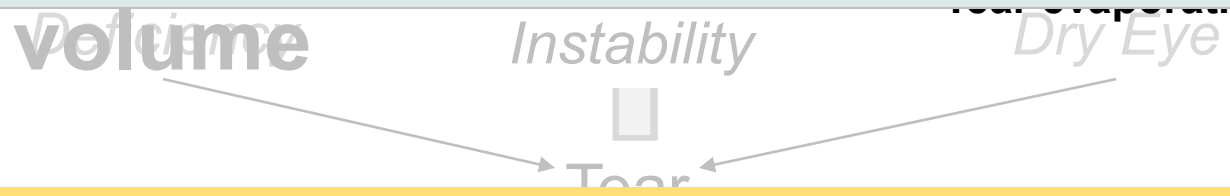


OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?  
Indeed it will—that is, unless steps are taken to normalize its chemical composition



that it's a semisolid means it can't even get out and onto the tear film.  
mainstay tx of 1) Application of heat to the eyelids to soften the  
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So the logic underpinning lid hygiene is:  
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

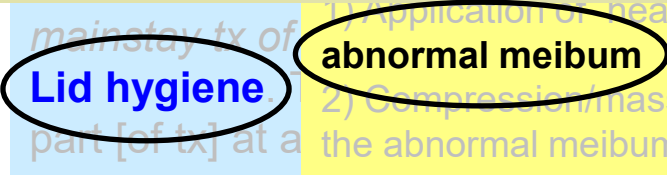
# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

- ?
- ?
- ?
- ?



1) Application of heat to the eyelids to soften the abnormal meibum  
2) Compression/massage of the lid margin to express the abnormal meibum

1) Increase

So the logic underpinning lid hygiene is:  
--Step 1: Liquify the semisolid abnormal meibum clogging the glands  
--Step 2: Express the now-liquefied abnormal meibum from the glands



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



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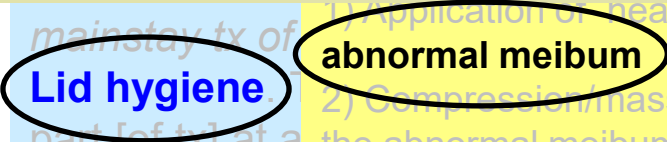
What steps/interventions can be taken in this regard?

- Topical [redacted]
- Topical [redacted]
- ?
- ?

that it's a semisolid means it can't even get out and onto the tear film.

mainstay tx of 1) Application of heat to the eyelids to soften the abnormal meibum

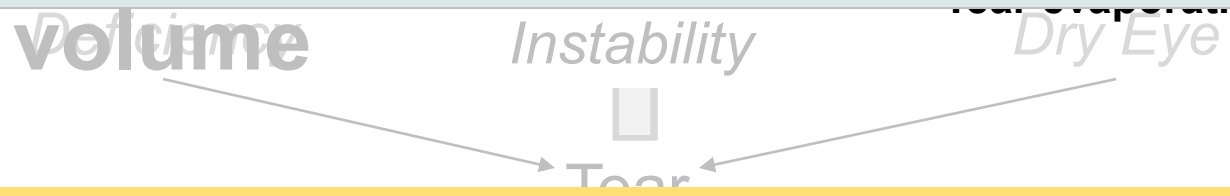
2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum



1) Increase

So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

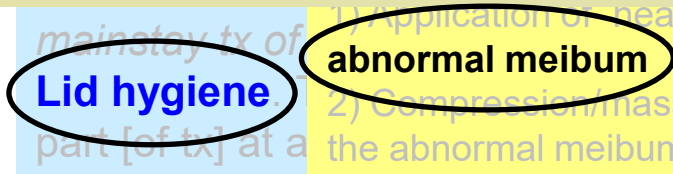
# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?  
Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

- Topical abx
- Topical steroids
- ?
- ?



1) Application of heat to the eyelids to soften the abnormal meibum  
2) Compression/massage of the lid margin to express part [of the] abnormal meibum

1) Increase volume

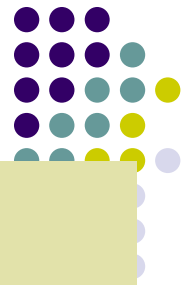
So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

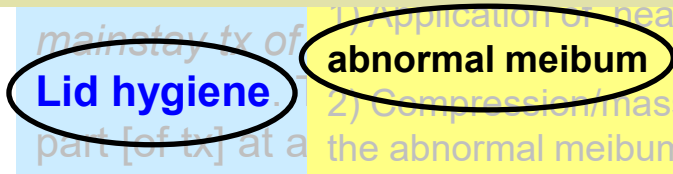
# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

- Topical abx
- Topical steroids
- PO [redacted]
- PO [redacted]

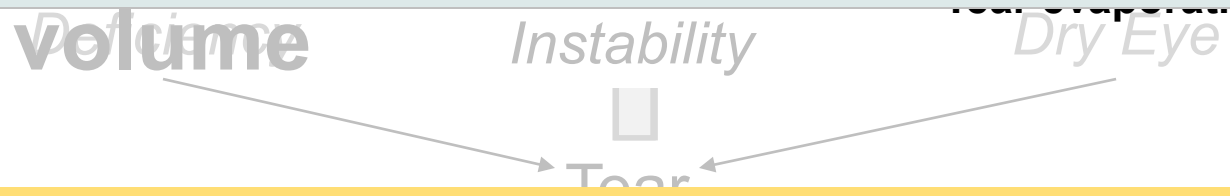


1) Application of heat to the eyelids to soften the abnormal meibum  
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1) Increase volume

So the logic underpinning lid hygiene is:

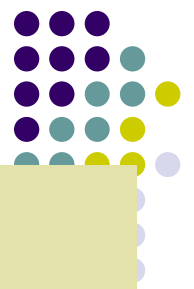
- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?  
 Indeed it will—that is, unless steps are taken to normalize its chemical composition

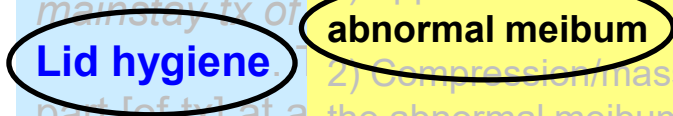
What steps/interventions can be taken in this regard?

- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

that it's a semisolid means it can't even get out and onto the tear film.

mainstay tx of 1) application of heat to the eyelids to soften the abnormal meibum

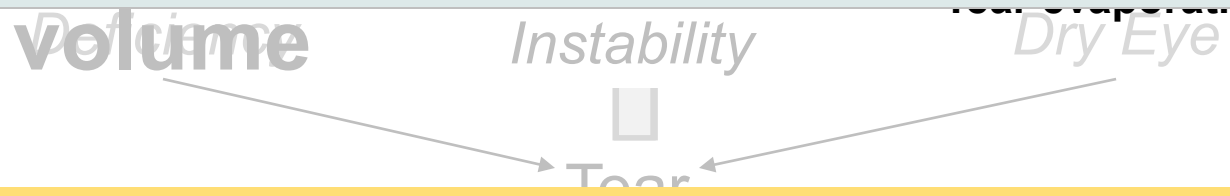
2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum



1) Increase

So the logic underpinning lid hygiene is:

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- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining part of the abnormal meibum still be there? Indeed it will—this is why the composition of the tear film is so important.

What steps/interventions are there?

- Topical steroids
- PO tetracyclines
- PO O3FA

Why is bacterial load diminution helpful?

**Topical abx**

mainstay tx of **abnormal meibum**

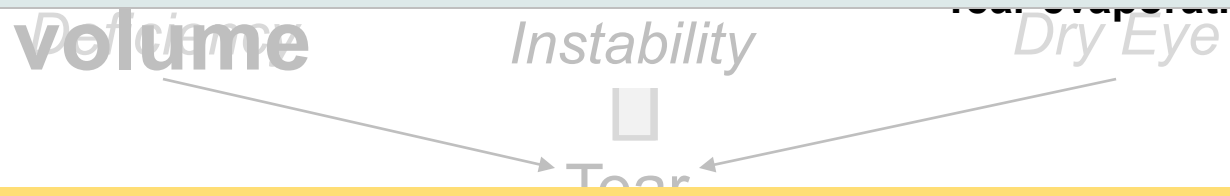
Lid hygiene.

1) Application of heat to the eyelids to soften the abnormal meibum  
 2) Compression/massage of the lid margin to express the abnormal meibum

1) Increase volume

So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining part of the abnormal meibum still be there? Indeed it will—

**Why is bacterial load diminution helpful?**  
 Bacterial        play an important role in altering meibum's chemical composition

**Topical abx**

--Topical steroid  
 --PO tetracycline  
 --PO O3FA

**abnormal meibum**

**Lid hygiene.**

1) Application of heat to the eyelids to soften the abnormal meibum  
 2) Compression/massage of the lid margin to express the abnormal meibum

1) Increase

So the logic underpinning lid hygiene is:

--Step 1: Liquify the semisolid abnormal meibum clogging the glands  
 --Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining part of the meibum gland be left behind? Indeed it will—this is why the composition of the meibum gland is important. The point of the abnormal meibum gland is a bit like the fact that it's a semisolid means it can't even get out and onto the tear film.

**Why is bacterial load diminution helpful?**  
Bacterial lipases play an important role in altering meibum's chemical composition

**Topical abx**

- Topical steroid
- PO tetracycline
- PO O3FA

**abnormal meibum**  
1) Application of heat to the eyelids to soften the meibum  
2) Compression/massage of the lid margin to express the abnormal meibum

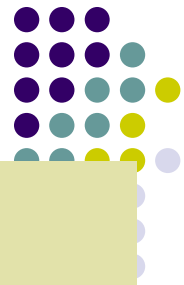
1) Increase

So the logic underpinning lid hygiene is:  
--**Step 1:** Liquify the semisolid abnormal meibum clogging the glands  
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining part of the abnormal meibum still be there? Indeed it will—

**Why is bacterial load diminution helpful?**  
 Bacterial lipases play an important role in altering meibum's chemical composition. **Reduce the bacterial load → reduce the lipase load → reduce the rate and degree of meibum alteration.**

**Topical abx**

--Topical steroid  
 --PO tetracycline  
 --PO O3FA

**abnormal meibum**

**Lid hygiene.**

1) Application of heat to the eyelids to soften the abnormal meibum  
 2) Compression/massage of the lid margin to express the abnormal meibum

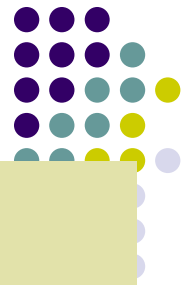
1) Increase

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining part of the abnormal meibum still be there? Indeed it will—

*Why is bacterial load diminution helpful?*  
 Bacterial lipases play an important role in altering meibum's chemical composition. Reduce the bacterial load → reduce the lipase load → reduce the rate and degree of meibum alteration.

**Topical abx**  
*Which topical abx is preferred for this?*

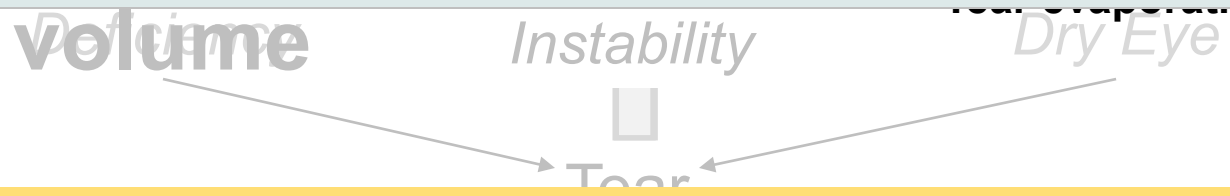
--PO tetracycline  
 --PO O3FA

*mainstay tx of*  
**Lid hygiene.** **abnormal meibum**

1) Application of heat to the eyelids to soften the part [of tx] at a the abnormal meibum  
 2) Compression/massage of the lid margin to express

1) Increase

So the logic underpinning lid hygiene is:  
 --Step 1: Liquify the semisolid abnormal meibum clogging the glands  
 --Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



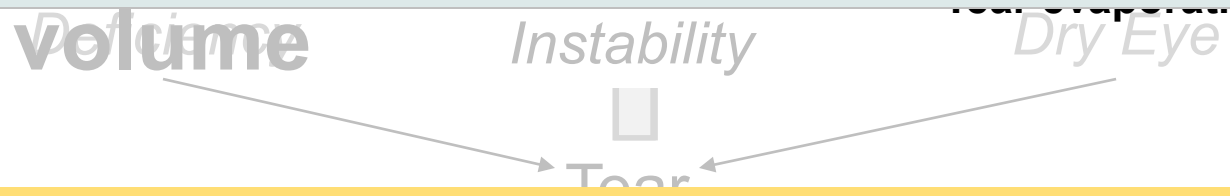
OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining...  
Indeed it will—t...  
What steps/inte...  
--Topical steroi...  
--PO tetracyclir...  
--PO O3FA

**Why is bacterial load diminution helpful?**  
Bacterial lipases play an important role in altering meibum's chemical composition. **Reduce the bacterial load**→reduce the **lipase load**→reduce the rate and degree of meibum alteration.  
**Which topical abx is preferred for this?**  
Azithromycin

**Topical abx**

**abnormal meibum**  
mainstay tx of...  
**Lid hygiene.**  
part [of tx] at a...  
1) Application of heat to the eyelids to soften the...  
2) Compression/massage of the lid margin to express the abnormal meibum

1) Increase...  
So the logic underpinning lid hygiene is:  
--**Step 1:** Liquify the semisolid abnormal meibum clogging the glands  
--**Step 2:** Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition

- What steps/interv...
- Topical abx
- Topical steroids
- PO tetracyclines**
- PO C3FA

PO tetracyclines in the pt is already on topical azithromycin— isn't that redundant?

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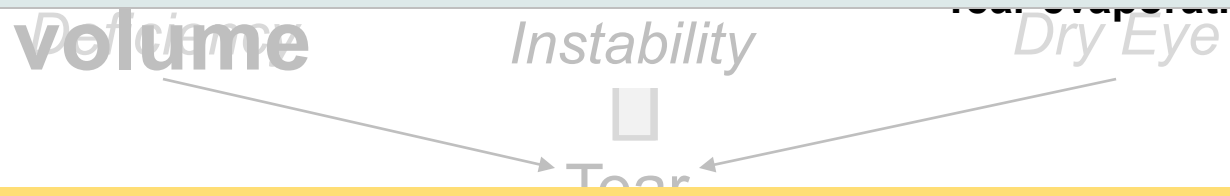
**Lid hygiene.**

**abnormal meibum**

2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum

1) Increase

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**



# Dry Eye Syndrome



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What steps/interv...

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- PO tetracyclines**
- PO C3FA

PO tetracyclines in the pt is already on topical azithromycin— isn't that redundant? You'd think so, but no—in MGD management, tetracyclines act primarily as an [redacted]

**Lid hygiene.** 2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum

1) Increase

So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions?

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- Topical steroids
- PO tetracyclines**
- PO C3FA

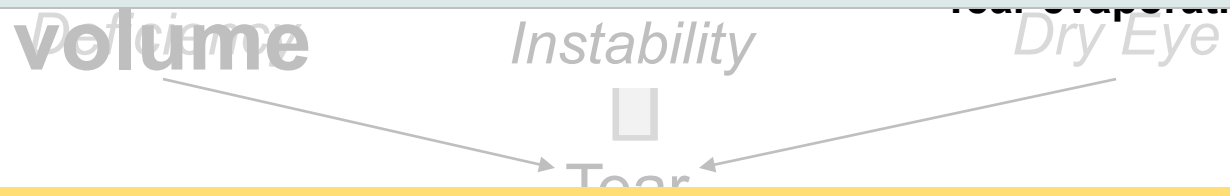
PO tetracyclines in the pt is already on topical azithromycin— isn't that redundant? You'd think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory

**Lid hygiene.** 2) Compression/massage of the lid margin to express the abnormal meibum

1) Increase volume

So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition

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- Topical steroids
- PO tetracyclines
- PO CSFA

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 What two anti-inflammatory properties do they possess?  
 --?  
 --?

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Lid hygiene. abnormal meibum  
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1) Increase  
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

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 --They reduce [redacted] release  
 --?

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# Dry Eye Syndrome



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What two anti-inflammatory properties do they possess?  
 --They reduce cytokine release  
 --?

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Lid hygiene. abnormal meibum  
 2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum

1) Increase

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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What two anti-inflammatory properties do they possess?  
 --They reduce cytokine release  
 --They inhibit **abb.** activity

**Lid hygiene.**

**abnormal meibum**

2) Compression/massage of the lid margin to express part [of tx] at a the abnormal meibum

1) Increase

So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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 You'd think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory

What two anti-inflammatory properties do they possess?  
 --They reduce cytokine release  
 --They inhibit MMP-9 activity

Lid hygiene. abnormal meibum

2) Compression/massage of the lid margin to express part [of tx] at the abnormal meibum

1) Increase

So the logic underpinning lid hygiene is:  
 --Step 1: Liquify the semisolid abnormal meibum clogging the glands  
 --Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion...  
Indeed it will—that is...  
Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

- What steps/interventions?
- Topical abx
- Topical steroids
- PO tetracyclines**
- PO C3FA

So the logic underpinning lid hygiene is:  
--Step 1: Liquify the semisolid abnormal meibum clogging the glands  
--Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**



# Dry Eye Syndrome

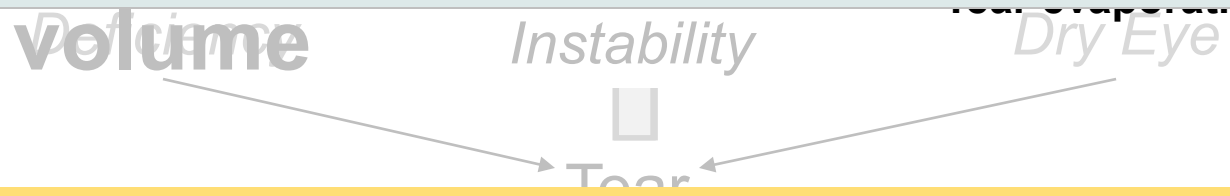


OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion...  
Indeed it will—that is...  
What steps/interventions...  
--Topical abx  
--Topical steroids  
**PO tetracyclines**  
--PO C3FA

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?  
It must be dosed frequently

So the logic underpinning lid hygiene is:  
--Step 1: Liquify the semisolid abnormal meibum clogging the glands  
--Step 2: Express the now-liquefied abnormal meibum from the glands

1) Increase



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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--PO C3FA

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?  
It must be dosed frequently, and it must be taken on an empty vs full stomach

So the logic underpinning lid hygiene is:  
--Step 1: Liquify the semisolid abnormal meibum clogging the glands  
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

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# Dry Eye Syndrome



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*Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?*  
It must be dosed frequently, and it must be taken on an empty stomach  
  
*What are the two alternatives that lack these limitations?*

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*How long is a typical course of tx?*

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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What steps/interventions?

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It must be dosed frequently, and it must be taken on an empty stomach

*What are the two alternatives that lack these limitations?*  
Minocycline and doxycycline

*How long is a typical course of tx?*  
4-6 days?  
weeks?  
months?  
years? maybe a little longer

1) Increase

So the logic underpinning lid hygiene is:

- Step 1: Liquify the semisolid abnormal meibum clogging the glands
- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

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*How long is a typical course of tx?*  
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So the logic underpinning lid hygiene is:  
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**



# Dry Eye Syndrome



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Minocycline and doxycycline

*How long is a typical course of tx?*  
4-6 weeks, maybe a little longer

*Is it considered appropriate to repeat the course if the initial response was less than hoped-for?*

So the logic underpinning lid hygiene is:

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- Step 2: Express the now-liquefied abnormal meibum from the glands



**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

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4-6 weeks, maybe a little longer

*Is it considered appropriate to repeat the course if the initial response was less than hoped-for?*  
Yes

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massaging the remaining portion of the gland. Indeed it will—that is, the gland will be able to produce more tears. What steps/interventions are there? --Topical abx --Topical steroids --PO tetracyclines --PO G3FA

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones? --? --? --? --? --? --?

Is it considered appropriate to repeat the course if the initial response was less than hoped-for? Yes

1) Increase

So the logic underpinning lid hygiene is: --Step 1: Liquify the semisolid abnormal meibum clogging the glands --Step 2: Express the now-liquefied abnormal meibum from the glands



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



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--Photosensitization  
--?  
--?  
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--?

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
Yes

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



OK, so heat and massaging the remaining portion of the eyelid. Indeed it will—that is, the meibomian glands. What steps/interventions are there? --Topical abx --Topical steroids --PO tetracyclines --PO C3FA

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones? --Photosensitization (pts should be instructed to avoid sun exposure) --? --? --? --? --? --?

**PO tetracyclines**

Is it considered appropriate to repeat the course if the initial response was less than hoped-for? Yes

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Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?  
--Photosensitization (pts should be instructed to avoid sun exposure)  
--GI upset  
--?  
--?  
--?

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
Yes

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So the logic underpinning lid hygiene is:  
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



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--Photosensitization (pts should be instructed to avoid sun exposure)  
--GI upset  
--Potentiation of effect in certain [redacted] meds  
--?  
--?

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
Yes

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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**



# Dry Eye Syndrome



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**PO tetracyclines**

Is it considered appropriate to repeat the course if the initial response was less than hoped-for? Yes

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So the logic underpinning lid hygiene is: --Step 1: Liquify the semisolid abnormal meibum clogging the glands --Step 2: Express the now-liquefied abnormal meibum from the glands



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--Photosensitization (pts should be instructed to avoid sun exposure)  
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--Potentiation of effect in certain anticoagulant meds (classic example: warfarin )  
--Reduction in effectiveness of two words  
--?

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
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--Potentiation of effect in certain anticoagulant meds (classic example: warfarin )  
--Reduction in effectiveness of oral contraceptives  
--?

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Yes

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--Reduction in effectiveness of oral contraceptives  
--Teeth [redacted] in children

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
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--Reduction in effectiveness of oral contraceptives  
--Teeth discoloration in children  
  
Can the tetracyclines be used during pregnancy?

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
Yes

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--Reduction in effectiveness of oral contraceptives  
--Teeth discoloration in children  
  
Can the tetracyclines be used during pregnancy? *Breastfeeding?*  
No

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
Yes

1) Increase

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--Teeth discoloration in children  
  
Can the tetracyclines be used during pregnancy? *Breastfeeding?*  
No. **No.**

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?  
Yes

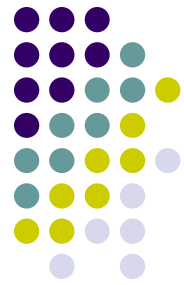
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**With regards to treating DES—there are three obvious interdiction points in its pathogenesis:**

# Dry Eye Syndrome



Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects **Inflammatory cytokine release**

What class of topical med is most effective in controlling ocular-surface inflammation?

poration

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



*3) Prevent cytokine release and/or mitigate their effects*

**Inflammatory cytokine release**



*What class of topical med is most effective in controlling ocular-surface inflammation?  
Steroids*

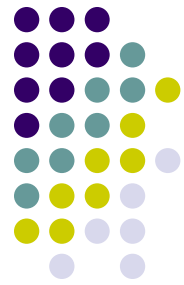
poration



Tear

*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects **Inflammatory cytokine release**

What class of topical med is most effective in controlling ocular-surface inflammation?  
Steroids

So why don't we keep all DES pts on them?

poration

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



*3) Prevent cytokine release and/or mitigate their effects*

**Inflammatory cytokine release**



What class of topical med is most effective in controlling ocular-surface inflammation?  
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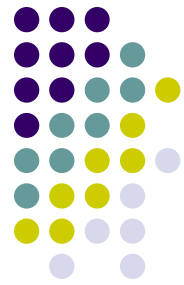
So why don't we keep all DES pts on them?  
Because of their terrible side-effect profile

poration



*With regards to treating DES—there are three obvious interdiction points in its pathogenesis:*

# Dry Eye Syndrome



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So why don't we keep all DES pts on them?  
Because of their terrible side-effect profile, ie, development of

poration

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



3) Prevent cytokine release and/or mitigate their effects **Inflammatory cytokine release**



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So why don't we keep all DES pts on them?  
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poration



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



3) Prevent cytokine release and/or mitigate their effects

**Inflammatory cytokine release**



What class of topical med is most effective in controlling ocular-surface inflammation?  
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So why don't we keep all DES pts on them?  
Because of their terrible side-effect profile, ie, development of cataracts, increased

abb.

poration



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



3) Prevent cytokine release and/or mitigate their effects **Inflammatory cytokine release**



What class of topical med is most effective in controlling ocular-surface inflammation?  
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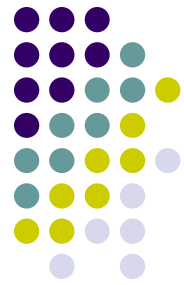
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poration



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

# Dry Eye Syndrome



Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects **Inflammatory cytokine release**

What class of topical med is most effective in controlling ocular-surface inflammation?  
Steroids

So why don't we keep all DES pts on them?  
Because of their terrible side-effect profile, ie, development of cataracts, increased IOP, and compromised ocular-surface

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

poration

# Dry Eye Syndrome



Decreased aqueous production



Neural reflex arc disruption



3) Prevent cytokine release and/or mitigate their effects **Inflammatory cytokine release**



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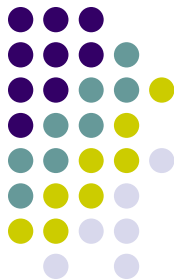
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It can take a l-o-n-g time for their effects to kick in—weeks (if you're lucky) to months (probably more typical). During the ramp-up period, compliance may become an issue as the pt gives up in frustration.

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Inflammatory cytokine release

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**Why does it take so long for these drugs to reach full effect?**

**lease**

Why are steroids contraindicated in the management of DES?  
Steroids

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It's probably related to the length of the T-cell life cycle (~120 days)

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What about steroids for ocular-surface inflammation?  
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Cyclosporine has three measurable effects on the ocular surface—  
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--?  
--?

How do they work  
They interfere with

What's the main dr  
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r-surface inflammation?

poration



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

Two steroid-sparing  
-- Cyclosporine  
-- Leflunomide

Cyclosporine has three measurable effects on the ocular surface—  
what are they?

- Reduced T-cell numbers
- Increased conjunctival numbers
- ?

two words

How do they work  
They interfere with

What's the main drug  
It can take a long time for their effects to kick in

... weeks (if you're lucky), to months (probably more as the pt gives up in frustration).

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corrosion

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Cyclosporine has three measurable effects on the ocular surface—  
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- Reduced T-cell numbers
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- Increased conjunctival goblet cell numbers
- Increased two-words production

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What potential ocular side effects are concerning?

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release

ocular-surface inflammation?

tear evaporation



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--Lifitegrast

Cyclosporine has three measurable effects on the ocular surface—  
what are they?

- Reduced T-cell numbers
- Increased conj goblet cell numbers
- Increased aqueous-tear production

What potential ocular side effects are concerning?

It has none (other than stinging)

How do they work  
They interfere with

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lease

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poration

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- Increased conj goblet cell numbers
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What potential ocular side effects are concerning? **Systemic?**  
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How do they work  
They interfere with

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- Increased aqueous-tear production

What potential ocular side effects are concerning? **Systemic?**

It has none (other than stinging). **It has no systemic side effects.**

How do they work  
They interfere with

What's the main dr  
It can take a l-o-n-g

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Cyclosporine has three measurable effects on the ocular surface—

**Lifitegrast**

How does lifitegrast reduce T-cell activity?

How do they work?  
They interfere with

What's the main effect?  
It can take a long

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release

ocular-surface inflammation?

corruption

T-cell

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



Two steroid-sparing  
--Cyclosporine

**Lifitegrast**

How does lifitegrast reduce T-cell activity?

By inhibiting abb. binding

Cyclosporine has three measurable effects on the ocular surface—

How do they wo  
They interfere w

What's the main  
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Why does it take so long for these drugs to reach full effect?

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Two steroid-sparing  
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Cyclosporine has three measurable effects on the ocular surface—

**Lifitegrast**

**How does lifitegrast reduce T-cell activity?  
By inhibiting ICAM-1 binding**

How do they wo  
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What's the main  
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Tear

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:



**Lifitegrast**

How does lifitegrast reduce T-cell activity?  
By inhibiting ICAM-1 binding

We covered it earlier in the slide-set, but remind me:  
What role does ICAM-1 play in the pathophysiology of DES?

Why does it take so long for these drugs to reach full effect?  
It's probably related to the length of the T-cell life cycle (~120 days)

What can be done to bridge the gap between commencement of therapy and onset of symptom relief?

A short course of topical steroids is ideal for this

Steroids

So why don't we keep all DES pts on them?  
Because of their terrible side-effect profile, ie, development of cataracts, increased IOP, and compromised ocular-surface immunity

So steroids are verboten in the management of DES?  
Not at all—it's just that they must be used judiciously (more shortly)

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

release

ocular-surface inflammation?

corrosion



**Lifitegrast**

*How does lifitegrast reduce T-cell activity?  
By inhibiting ICAM-1 binding*

*We covered it earlier in the slide-set, but remind me:  
What role does ICAM-1 play in the pathophysiology of DES?  
It promotes/facilitates T-cell migration to the ocular surface and lacrimal gland*

*Why does it take so long for these drugs to reach full effect?  
It's probably related to the length of the T-cell life cycle (~120 days)*

*What can be done to bridge the gap between commencement of therapy and onset of symptom relief?*

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**Steroids**

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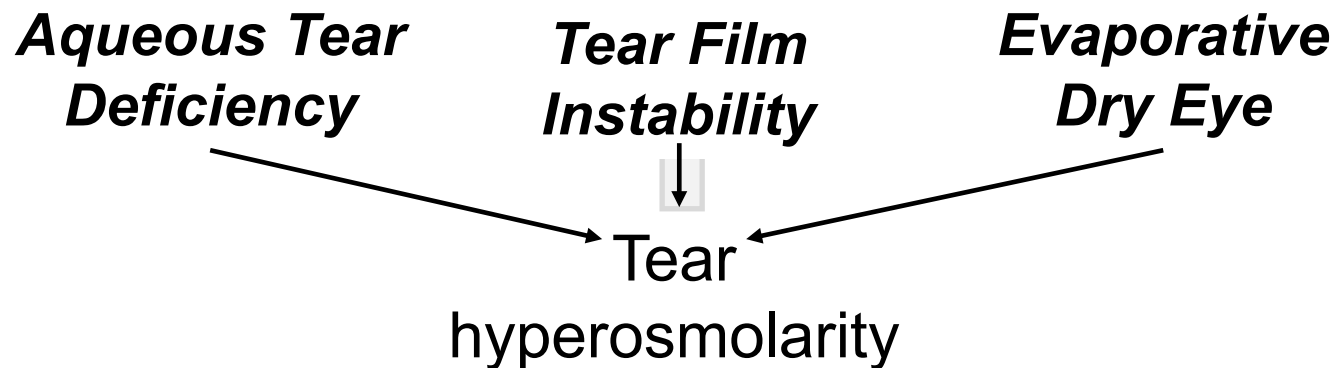
**lease**

*poration*

## Dry Eye Syndrome



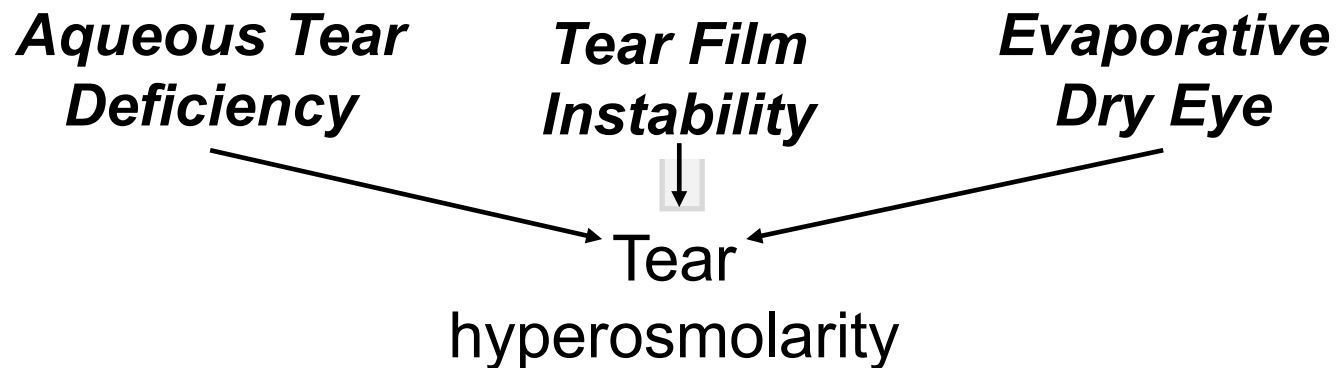
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## Dry Eye Syndrome



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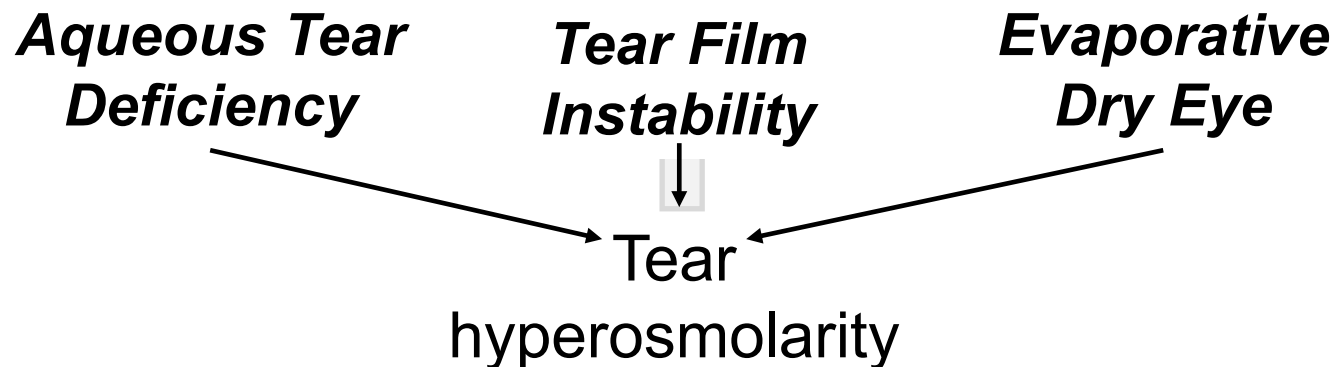


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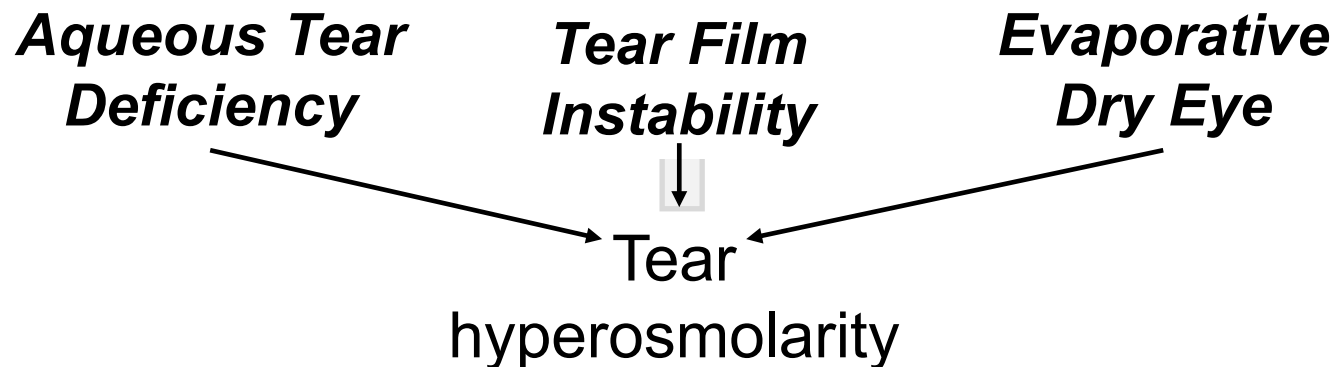
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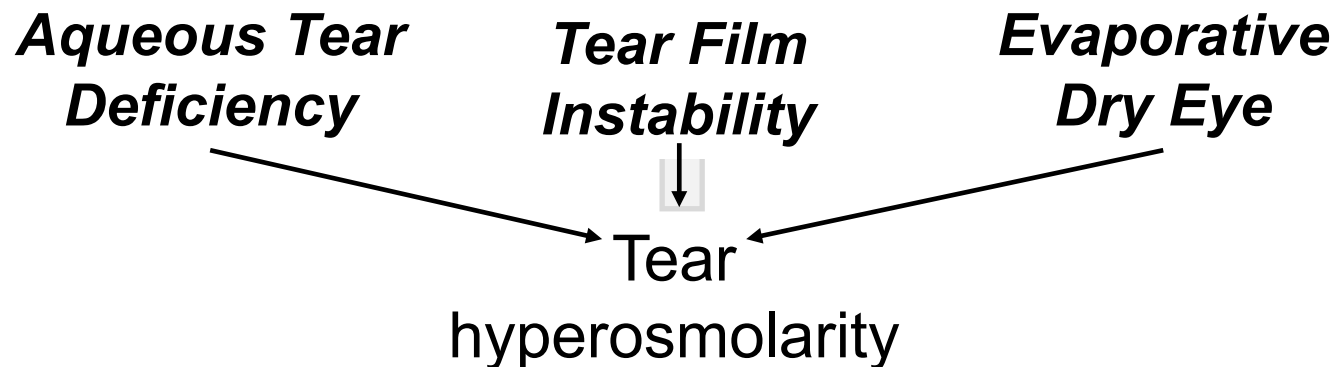
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## Dry Eye Syndrome

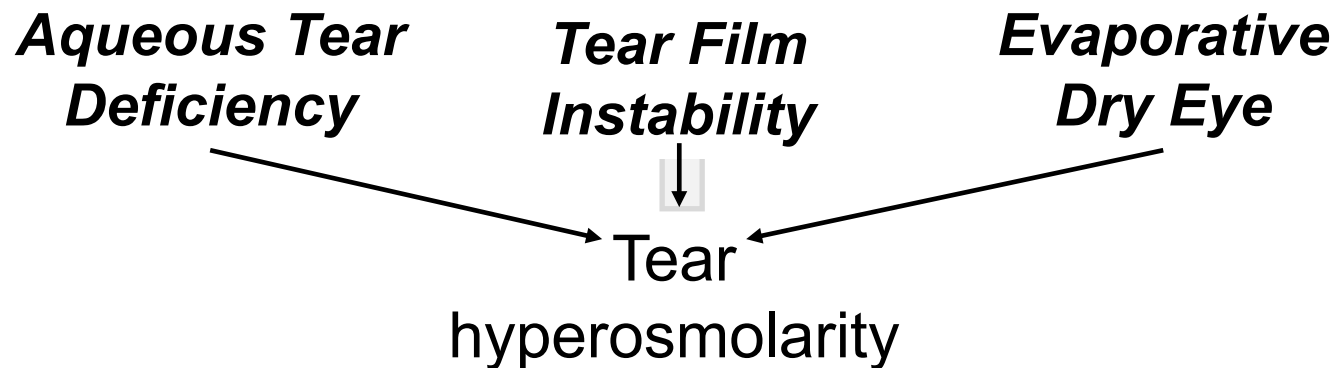


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**Punctal occlusion**



## Dry Eye Syndrome



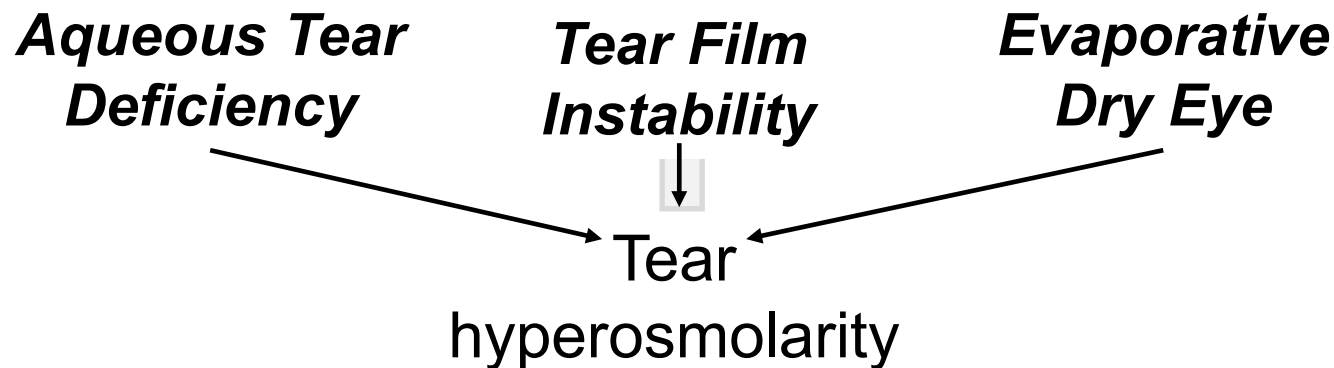
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*Why must punctal occlusion be used with caution in ATD pts with concurrent MGD?*



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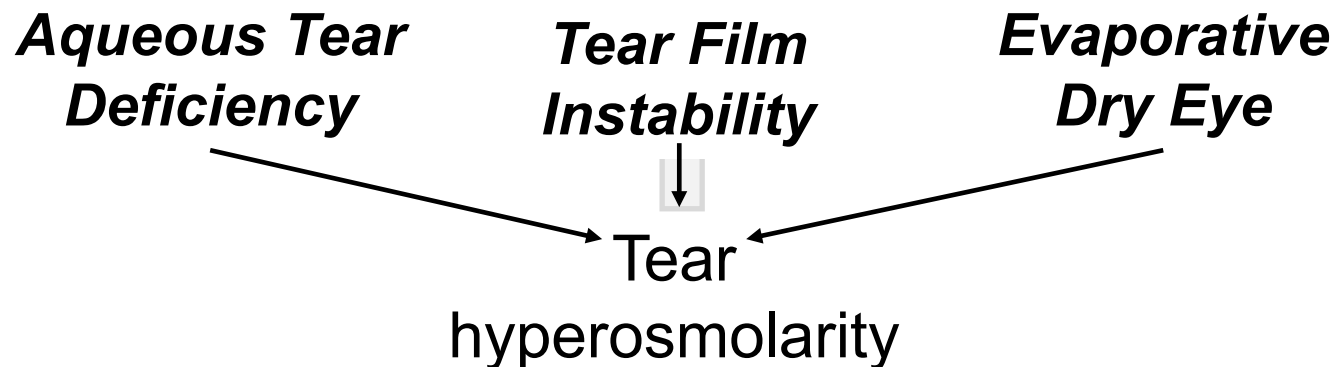
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Punctal occlusion

*Why must punctal occlusion be used with caution in ATD pts with concurrent MGD?*

Because in addition to increasing the amount of aqueous on the ocular surface (good), occlusion will also increase/maintain the proinflammatory abnormal meibum on the ocular surface (bad).



# Dry Eye Syndrome



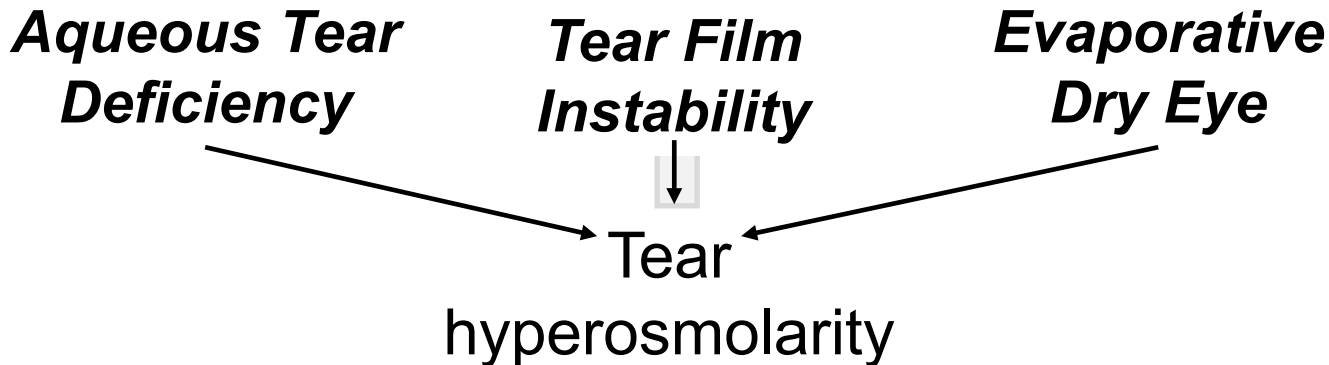
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**Remember when we said this?**



If a pt has both, what are the implications for management? Most interventions (ATs, anti-inflammatory meds, O3FA) are useful in both conditions. However, **Note: There is another complication induced by the use of punctal occlusion that we will cover later in the slide-set** with caution in Punctal occlusion

Why must punctal occlusion be used with caution in ATD pts with concurrent MGD? Because in addition to increasing the amount of aqueous on the ocular surface (good), occlusion will also increase/maintain the proinflammatory abnormal meibum on the ocular surface (bad). In general, you want to control the inflammatory component of a pt's DES before you occlude their puncta.

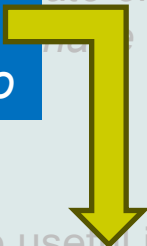
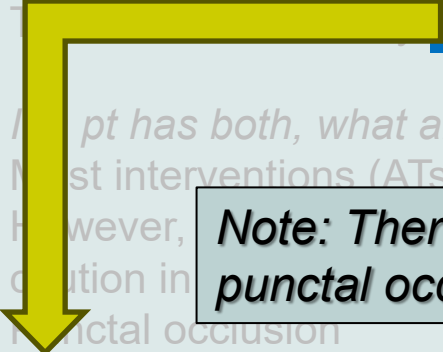


# Dry Eye Syndrome



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**Remember when we said this?  
This is what we were referring to**



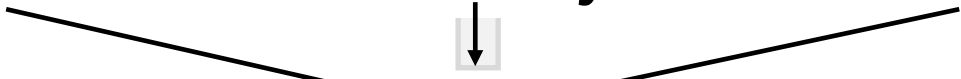
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**Aqueous Tear Deficiency**

**Tear Film Instability**

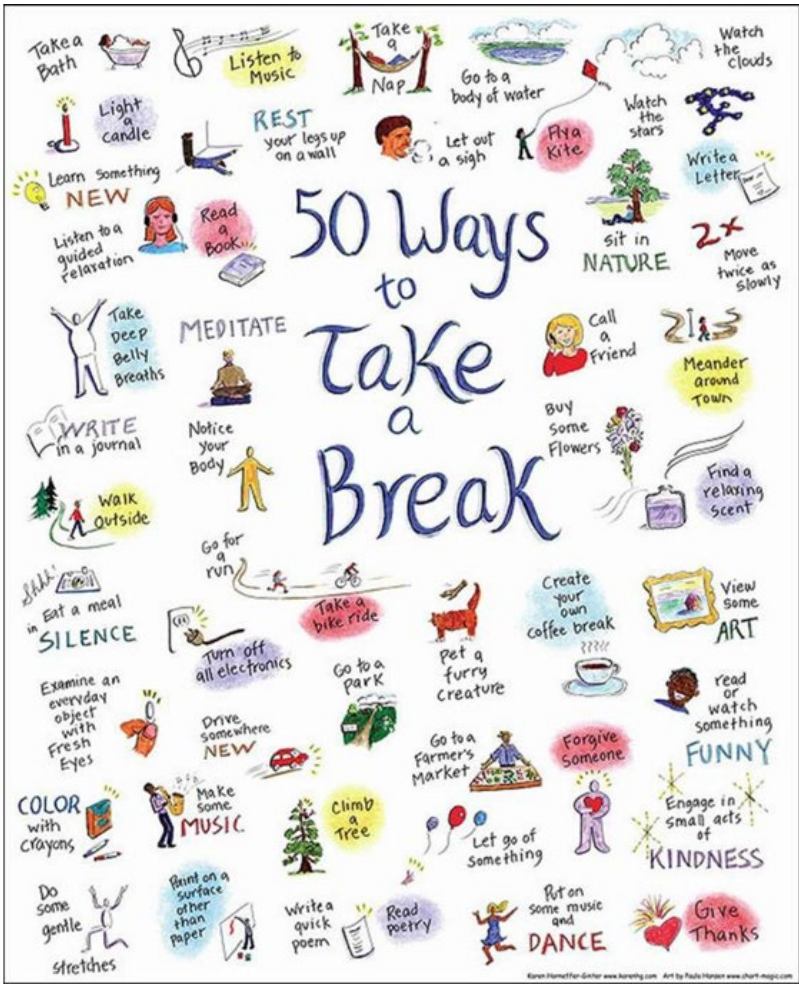
**Evaporative Dry Eye**



**Tear**

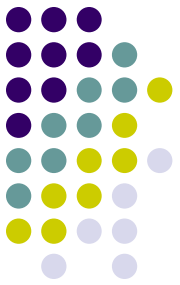
**hyperosmolarity**

# Dry Eye Syndrome



(This is a good point in the set to take a break)

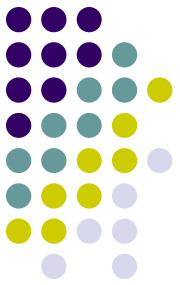
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- ?
- ?
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## Dry Eye Syndrome

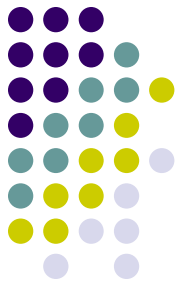


*Finally: The Cornea book discusses several conditions that mimic DES in their presentation—what are they?*

- Conjunctivochalasis
- SLK
- Floppy eyelid syndrome
- Nighttime lagophthalmos
- Parkinson's
- Mucous-membrane pemphigoid/OCP



# Dry Eye Syndrome



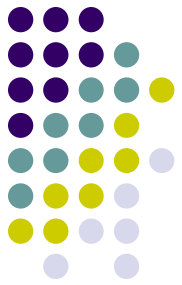
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**--Conjunctivochalasis**

--SI K  
--F  
--M  
--F  
--M

*Briefly, what is conj'chalasis?*

# Dry Eye Syndrome



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## **--Conjunctivochalasis**

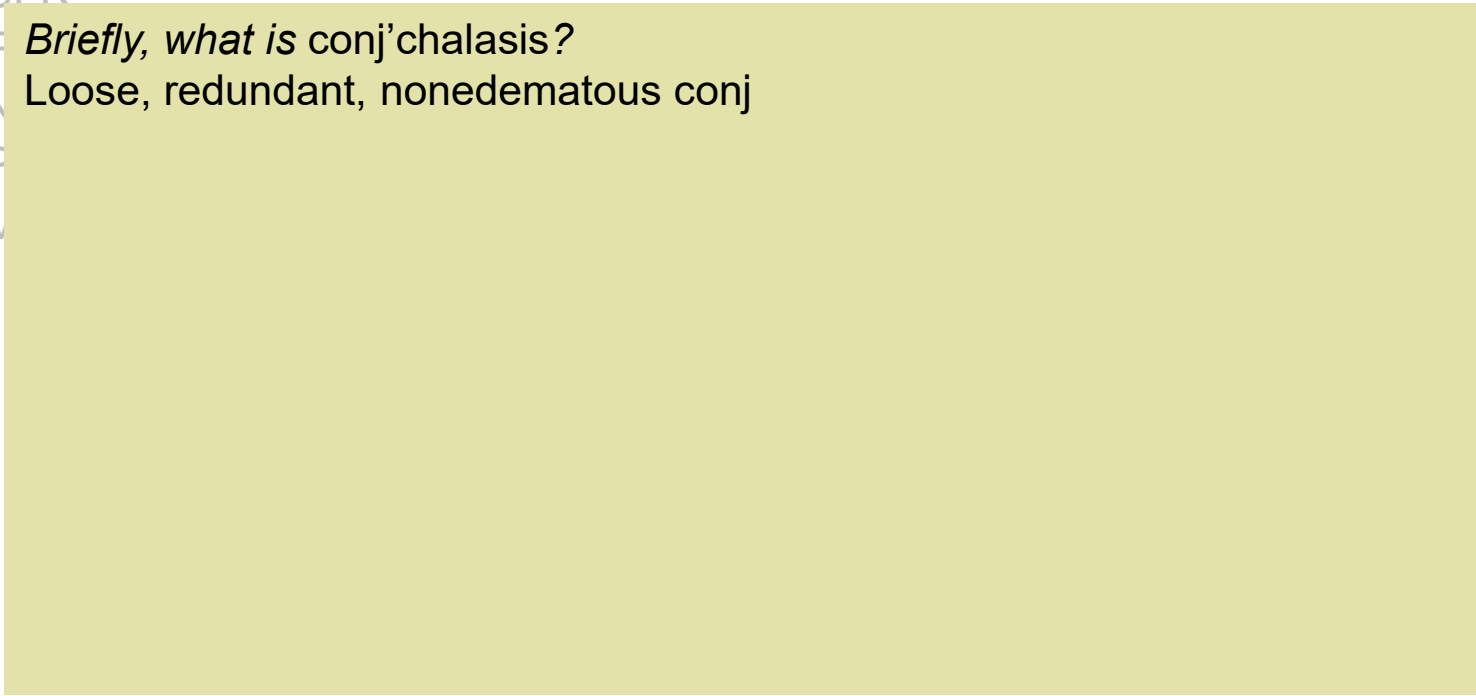
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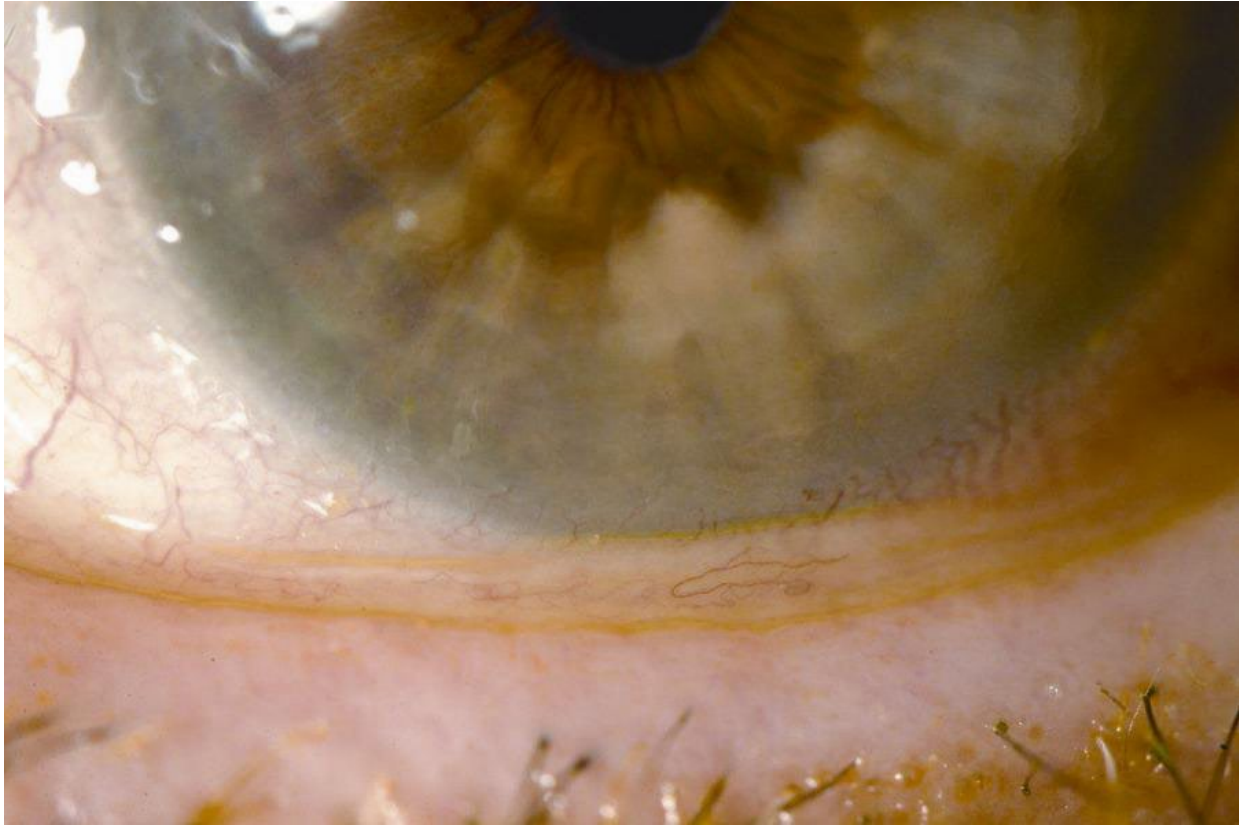
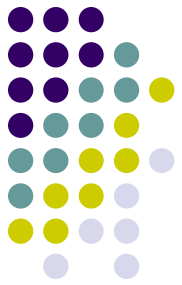
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# Dry Eye Syndrome



Conjunctivochalasis

# Dry Eye Syndrome



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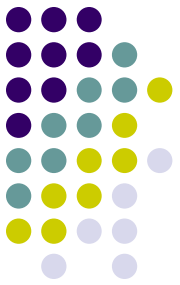
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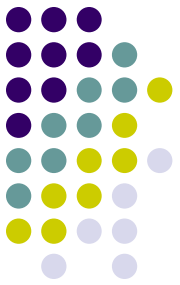
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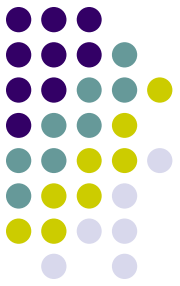
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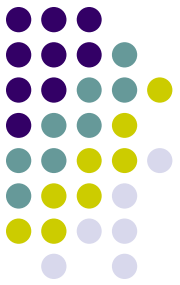
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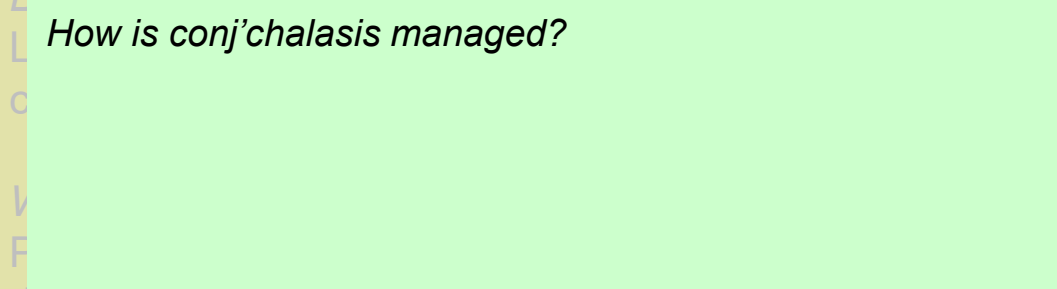
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(although one of the characteristics of conj'chalasis is that it  
--M V doesn't respond well to DES-tx maneuvers).

F *What is conj'chalasis?* It is a fold of conjunctiva that protrudes against the bulbar conjunctiva during blinking

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It's reasonable to start with ATs, antihistamines, steroids etc (although one of the characteristics of conj'chalasis is that it doesn't respond well to DES-tx maneuvers). Often, surgical intervention (in the form of thermal cicatrization or excision) to remove the redundant conj is required for resolution.

--F C

--M V

during blinking

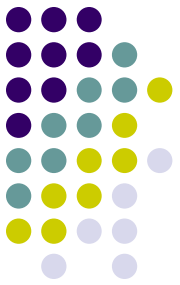
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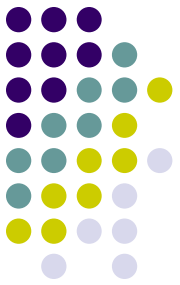
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--**SLK**

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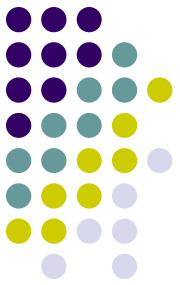
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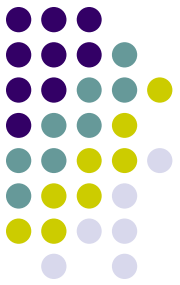
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*Is it common, or rare?*





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Yes, 

M
v F

 are 

s/w v
far

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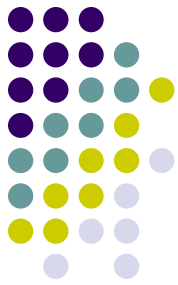
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*Is there a gender predilection?*

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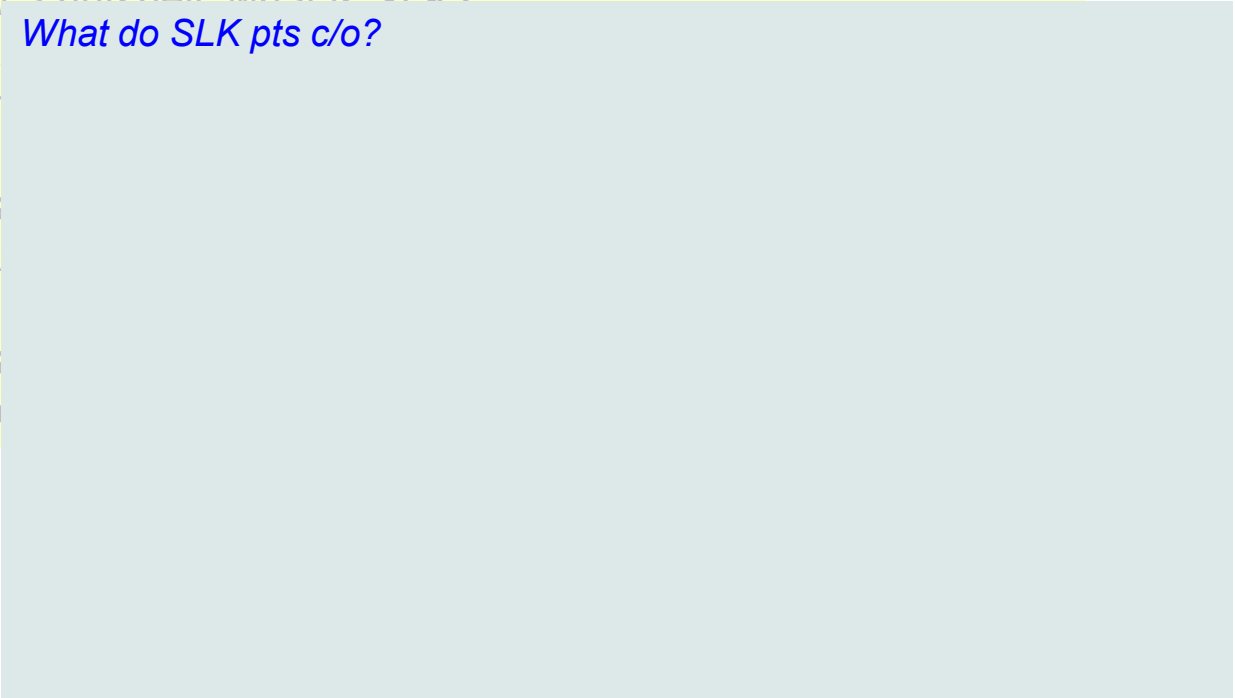
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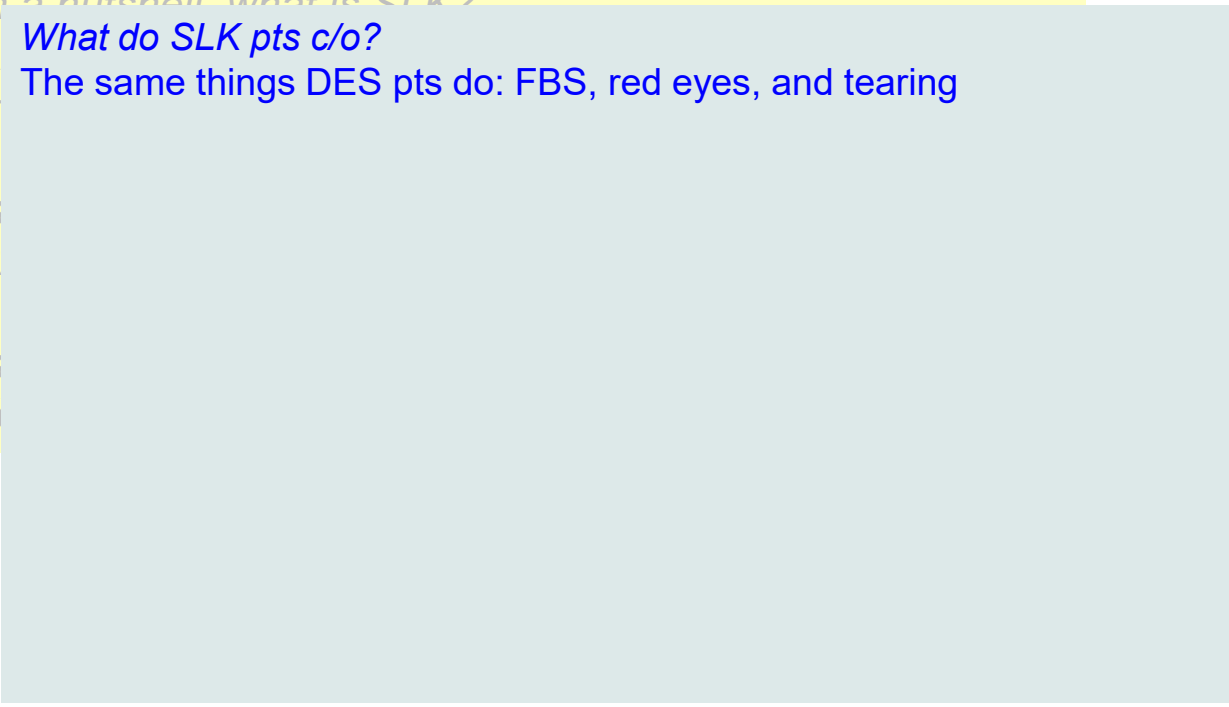
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Is

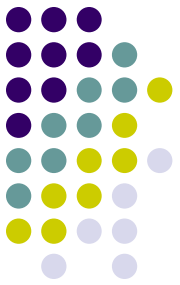
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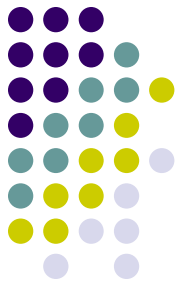
Is

Y

A large light blue rectangular area covering the lower half of the slide, likely representing a redacted section or a placeholder for an image.



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--Conjunctivochalasis

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-- *In a nutshell, what is SLK?*

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-- *SLK has three classic findings associated with the superior bulbar conj.*

Is *What are they?*

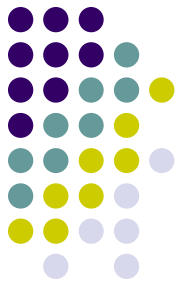
R --?

--?

Is --?

Y

# Dry Eye Syndrome



*Finally: The Cornea book discusses several conditions that mimic DES in their presentation—what are they?*

--Conjunctivochalasis

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R -- **vascular status**

--?

Is --?

Y

# Dry Eye Syndrome



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R --Injection

--?

Is --?

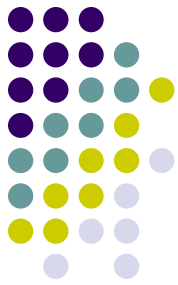
Y

# Dry Eye Syndrome



SLK: Superior conj injection

# Dry Eye Syndrome



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R --Injection

--It is **tautness**

Is --?

Y

# Dry Eye Syndrome



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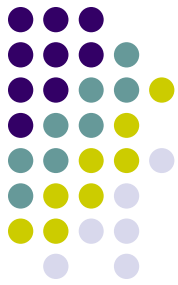
R --Injection

--It is redundant/loose

*Is --?*

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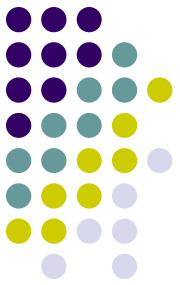
R --Injection

--It is redundant/loose

*Is* --It stains with   and/or

*Y*

# Dry Eye Syndrome



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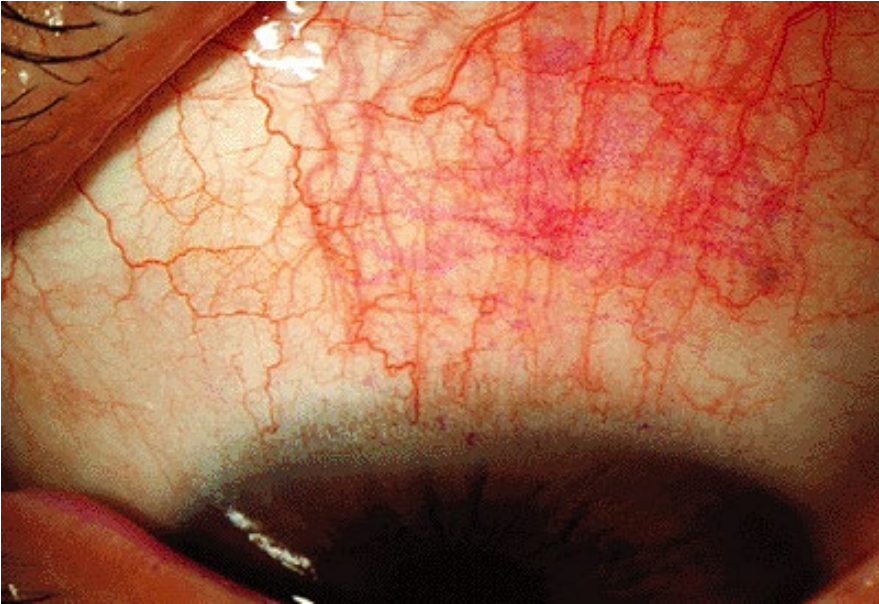
--It is redundant/loose

*Is --It stains with **rose bengal**, **lissamine green**, and/or **fluorescein***

*Y*



# Dry Eye Syndrome



Superior rose bengal staining



Superior lissamine green staining

SLK staining

# Dry Eye Syndrome



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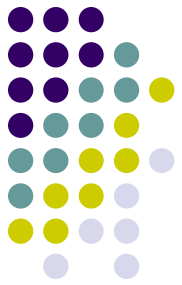
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Papillary reaction

# Dry Eye Syndrome



SLK: Superior tarsal conj papillary rxn

# Dry Eye Syndrome



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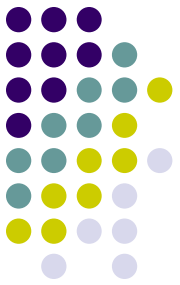
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Papillary reaction

*SLK has # classic cornea findings.*

# Dry Eye Syndrome



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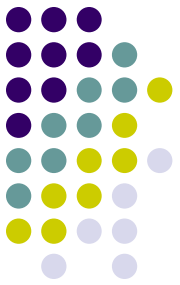
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*SLK has two classic cornea findings.*

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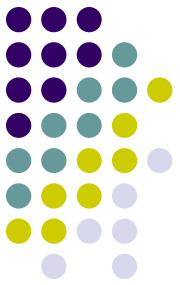
Papillary reaction

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--?

--?

# Dry Eye Syndrome



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-- *Papillary reaction*

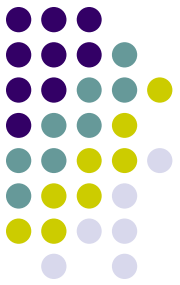
-- *SLK has two classic cornea findings. What are they?*

--Superior

--Superior



# Dry Eye Syndrome



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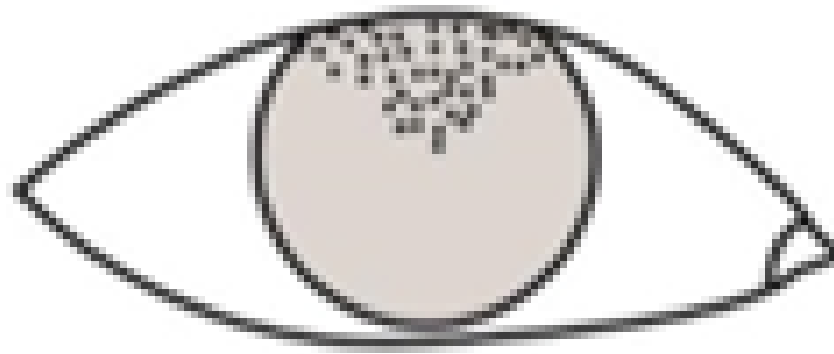
Papillary reaction

*SLK has two classic cornea findings. What are they?*

--Superior PEE/K

--Superior filaments

# Dry Eye Syndrome



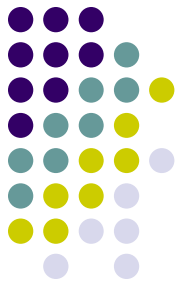
K staining pattern in SLK

# Dry Eye Syndrome



SLK: Superior corneal filaments

# Dry Eye Syndrome



*Finally: The Cornea book discusses several conditions that mimic DES in their presentation—what are they?*

--Conjunctivochalasis

--**SLK**

*In a nutshell, what is SLK?*

*A: T*

*So, SLK pts have irritated and redundant superior **bulbar conj**, irritated superior **tarsal conj**, and superior **corneal abnormalities**. What's the mechanism for all this?*

*S*

*Is V*

*R*

*--*

*Is*

*Y*

*S*

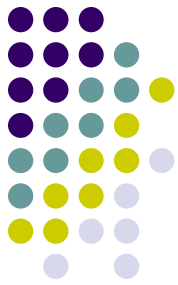
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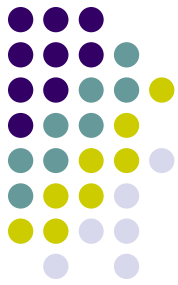
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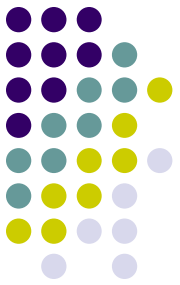
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*S* **The **mechanical theory** is the most widely accepted.** According to this theory, the superior lid is too tightly apposed to the globe, and the resulting excessive contact and rubbing produces the signs/symptoms of SLK.

*Is*

*Y*

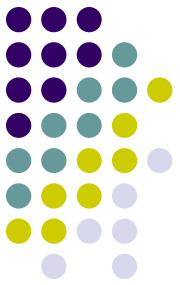
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*So, SLK pts have irritated and redundant superior **bulbar conj**, irritated superior **tarsal conj**, and superior **corneal abnormalities**. What's the mechanism for all this?*

*The **mechanical theory** is the most widely accepted. According to this theory, the superior lid is too tightly apposed to the globe, and the resulting excessive contact and rubbing produces the signs/symptoms of SLK.*

*Why do SLK pts have overly tight superior lids?*

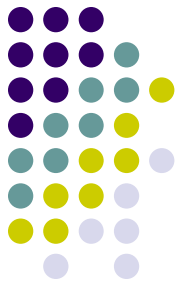
*SLK has two classic cornea findings—what are they?*

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# Dry Eye Syndrome



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*S* *The **mechanical theory** is the most widely accepted. According to this theory,*  
*Is V* *the superior lid is too tightly apposed to the globe, and the resulting excessive*  
*R* *contact and rubbing produces the signs/symptoms of SLK.*

*Is* *Why do SLK pts have overly tight superior lids?*

*Y* *In many cases, because of concomitant **classic systemic assn.** producing orbital congestion*  
*S* *that forces the globes forward against the lids.*

*Papillary reaction*

*SLK has two classic cornea findings—what are they?*

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# Dry Eye Syndrome



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*Is* *Why do SLK pts have overly tight superior lids?*

*Y* *In many cases, because of concomitant thyroid dz producing orbital congestion*  
*S* *that forces the globes forward against the lids.*

*Papillary reaction*

*SLK has two classic cornea findings—what are they?*

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# Dry Eye Syndrome



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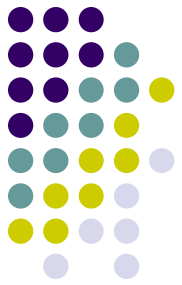
*Y* *In many cases, because of concomitant thyroid dz producing orbital congestion*  
*S* *that forces the globes forward against the lids. Check thyroid labs on all SLK pts!*

*Papillary reaction*  
*SLK has two classic cornea findings—what are they?*

--Superior PEE/K

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# Dry Eye Syndrome



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-- S T What are the two overarching goals in treating SLK? is?

-- Is V s --? ct

-- R -- a

--

-- Is -- V

-- Y Ir

-- S th

-- Pup

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--St

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# Dry Eye Syndrome



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-- R -- a --Reduce...

--

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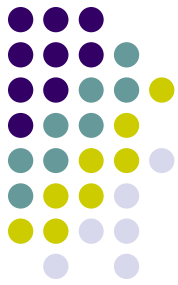
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-- Is V T --Reduce...surface inflammation

-- R --a --Reduce...friction between the superior bulbar conj and superior tarsal conj

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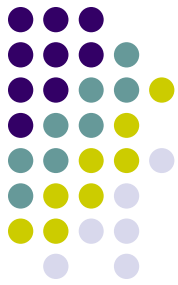
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-- S th --?

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SLK

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--Superior filaments

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-- S Ir --Topical anti-inflammatory meds

-- P th --Large (enough to cover the involved conj) diameter BCL

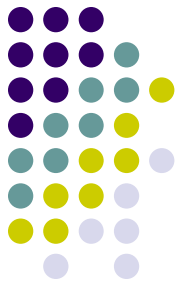
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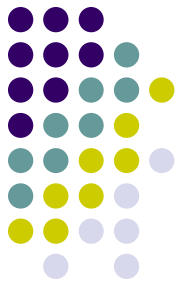
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# Dry Eye Syndrome



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--Conjunctivochalasis

--**SLK**

*In a nutshell, what is SLK?*

***For more on SLK, see slide-set K7***

*What are the two overarching goals in treating SLK?*

--Reduce...surface inflammation

--Reduce...friction between the superior bulbar conj and superior tarsal conj

*There are a number of medical treatment options. These include:*

--Preservative-free ATs

--Topical anti-inflammatory meds

--Large (enough to cover the involved conj) diameter BCL

*Is surgery ever indicated to resolve the redundant conj?*

It is indeed

--Superior filaments

*In a nutshell, what is floppy eyelid syndrome (FES)?*

*Finally: The Cornea*

*DES in their prese*

--Conjunctivochalaz

--SLK

--**Floppy eyelid syndrome**

--Nighttime lagoph

--Parkinson's

--Mucous-membra

*In a nutshell, what is floppy eyelid syndrome (FES)?*

A condition characterized by 1) upper-lid [redacted] and 2) chronic [redacted] of the ocular surface

*Finally: The Cornea*

*DES in their prese*

--Conjunctivochalaz

--SLK

--**Floppy eyelid syndrome**

--Nighttime lagoph

--Parkinson's

--Mucous-membra

*In a nutshell, what is floppy eyelid syndrome (FES)?*

A condition characterized by 1) upper-lid laxity and 2) chronic inflammation of the ocular surface

*Finally: The Cornea*

*DES in their presence*

--Conjunctivochalasis

--SLK

--**Floppy eyelid syndrome**

--Nighttime lagophthalmos

--Parkinson's

--Mucous-membrane

*In a nutshell, what is floppy eyelid syndrome (FES)?*

A condition characterized by 1) **upper-lid laxity** and 2) chronic inflammation of the ocular surface

*How can you tell if the UL is lax?*

*Finally: The Cornea*

*DES in their presence*

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*In a nutshell, what is floppy eyelid syndrome (FES)?*

A condition characterized by 1) **upper-lid laxity** and 2) chronic inflammation of the ocular surface

*How can you tell if the UL is lax?*

It will evert easily and dramatically with traction

*Finally: The Cornea*

*DES in their presence*

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--**Floppy eyelid syndrome**

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# Dry Eye Syndrome



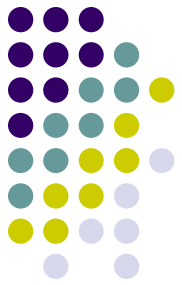
FES. Wow.

# Dry Eye Syndrome



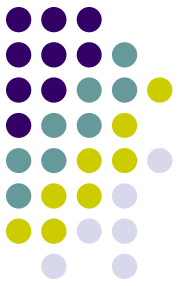
If you can't tell, that's an upper lid so lax it can be pinched like this

# Dry Eye Syndrome



FES. Note the fine two words (another common finding)

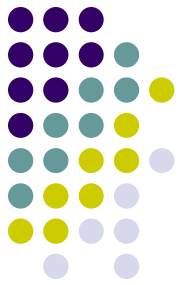
## Dry Eye Syndrome



FES. Note the fine papillary rxn (another common finding)

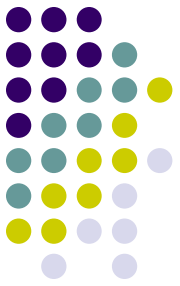


# Dry Eye Syndrome



This image demonstrates two words, another classic sign of FES

## Dry Eye Syndrome



This image demonstrates lash ptosis , another classic sign of FES

*In a nutshell, what is floppy eyelid syndrome (FES)?*

A condition characterized by 1) upper-lid laxity and 2) chronic inflammation of the ocular surface

*What do FES pts complain of?*

*Finally: The Cornea*

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*In a nutshell, what is floppy eyelid syndrome (FES)?*

A condition characterized by 1) upper-lid laxity and 2) chronic inflammation of the ocular surface

*What do FES pts complain of?*

FBS and mucous discharge that are worse in the

morning vs  
evening

*Finally: The Cornea*

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Obesity

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*How is FES managed initially?*

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Obesity

*How is FES managed initially?*

--Apply [redacted] to the involved eye(s) at qHS

--?

Finally: The Cornea

DES in their presence

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Obesity

*How is FES managed initially?*

--Apply ointment to the involved eye(s) at qHS, and

--Prevent eversion by either  the eye(s) or

Finally: The Cornea

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Obesity

*How is FES managed initially?*

--Apply ointment to the involved eye(s) at qHS, and

--Prevent eversion by either shielding the eye(s) or taping it/them shut

Finally: The Cornea

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*If FES fails to respond to the above, what's next?*

Surgical tightening of the lax upper lid(s)

Finally: The Cornea

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*If FES fails to respond to the above, what's next?*

Surgical tightening of the lax upper lid(s)

*With what potentially lethal systemic condition is FES strongly associated?*

*Finally: The Cornea*

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Surgical tightening of the lax upper lid(s)

*With what potentially lethal systemic condition is FES strongly associated?*

Obstructive sleep apnea

*Finally: The Cornea*

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*What is the main risk factor for FES? (It's systemic, not ocular.)*

Obesity

*How is FES managed initially?*

--Apply ointment to the involved eye(s) at qHS, and

--Prevent eversion by either shielding the eye(s) or taping it/them shut

*If FES fails to respond to the above, what's next?*

Surgical tightening of the lax upper lid(s)

*With what potentially lethal systemic condition is FES strongly associated?*

Obstructive sleep apnea. The BCSC states that all FES pts should be evaluated for OSA.

Finally: The Cornea

DES in their presence

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